THE NEW SYDENHAM SOCIETY.

INSTITUTED MDCCCLVIII.

VOLUME LXXVI.

LECTURES

ON

SURGICAL PATHOLOGY AND THERAPEUTICS.

A HANDBOOK FOR STUDENTS AND PRACTITIONERS.

BY

DR. THEODOR BILLROTH,
PROFESSOR OF SURGERY IN THE UNIVERSITY OF VIENNA.

TRANSLATED FROM THE EIGHTH EDITION.

VOL. II.

(WITH INDEX.)

LONDON:
THE NEW SYDENHAM SOCIETY.
MDCCCLXXVIII.

LONDON:
PRINTED BY J. E. ADLARD, BARTHOLOMEW CLOSE.

CONTENTS.

LECTURE XXIV.

CHAPTER XIII.

TRAUMATIC AND INFLAMMATORY COMPLICATIONS, WHICH MAY ACCIDENTALLY BEFALL WOUNDS. POISONED WOUNDS.

LECTURE XXV.

LECTURE XXVI.

II. GENERAL INCIDENTAL DISEASES WHICH MAY SUPERVENE UPON WOUNDS AND INFLAMMATION-NESTS. 1. WOUND- AND INFLAMMATION-FEVER; 2. SEPTIC FEVER AND SEPTICÆMIA; 3. SUPPURATIVE FEVER AND PYÆMIA.

LECTURE XXVII.

LECTURE XXVIII.

CHAPTER XIV.

ON CHRONIC INFAMMATION, ESPECIALLY OF THE SOFT PARTS.

LECTURE XXIX.

LECTURE XXX.

CHAPTER XV.

ON ULCERS.

LECTURE XXXI.

CHAPTER XVI.

ON CHRONIC INFLAMMATION OF THE PERIOSTEUM AND THE BONES AND ON NECROSIS.

LECTURE XXXII.

LECTURE XXXIII.

LECTURE XXXIV.

LECTURE XXXV.

APPENDIX TO CHAPTER XVI.

LECTURE XXXVI.

CHAPTER XVII.

ON CHRONIC INFLAMMATION OF THE JOINTS.

LECTURE XXXVII.

LECTURE XXXVIII.

APPENDIX.

LECTURE XXXIX.

APPENDIX I.

APPENDIX II.

LECTURE XL.

CHAPTER XVIII.

ON ANKYLOSIS; VARIETIES; ANATOMICAL CONDITIONS; DIAGNOSIS; TREATMENT; GRADUAL, FORCED EXTENSION, OPERATION.

LECTURE XLI.

CHAPTER XIX.

ON CONGENITAL MYO- AND NEUROPATHIC JOINT DEFORMITIES AND ON CICATRICIAL CONTRACTIONS. LOXARTHROSES.

LECTURE XLII.

CHAPTER XX.

ON VARICES AND ANEURISMS.

LECTURE XLIII.

CHAPTER XXI.

TUMOURS.

LECTURE XLIV.

LECTURE XLV.

LECTURE XLVI.

LFCTURE XLVII.

LECTURE XLVIII.

LECTURE XLIX.

LECTURE L.

CHAPTER XXII.

ON AMPUTATIONS, EXARTICULATIONS, AND RESECTIONS.

LECTURE XXIV.

CHAPTER XIII.

TRAUMATIC AND INFLAMMATORY COMPLICATIONS, WHICH MAY ACCIDENTALLY BEFALL WOUNDS. POISONED WOUNDS.

1 *Local diseases which may attack wounds and other inflammatory centres.* (1) *Progressive suppuration and diffuse sloughing of the subcutaneous cellular tissue.* (2) *Hospital gangrene.*
10 *Ulcerative muco-salivary diphtheritis. Ulcerative urinary diphtheritis.* (3) *Traumatic erysipelas.* (4) *Lymphangitis.*

Gentlemen,—Somewhat earlier on in my lectures, when speaking of traumatic inflammation, I stated it did not usually extend beyond the limits of the injury, and that if this sometimes appeared to be the case, it was due to the fact that we had overlooked the extent of the injury. I strongly maintain the truth of this remark. We have, however, added that very severe and progressive inflammation with sloughing may take place as the result of accidents (such as contused wounds) either immediately after the injury, or that secondary inflammations may develop even in wounds which are granulating, from causes which we also discussed. I must now point out to you that there is still another class of peculiar inflammatory and gangrenous processes, which attack wounds and lead to severe and, for the most part, pyrexial constitutional diseases. These latter may even come on without any appreciable change in the condition of the wound. Lastly, substances may enter an already existing wound, or even at the time of its occurrence (as from the bite of a poisonous or diseased animal), which may lead to severe local inflammation or general blood poisoning. We will first of all speak of the local manifestations which, as

complications, may befall a wound or a local inflammation, brought on by other and independent causes.

1. *Local diseases which may complicate wounds and other circumscribed inflammations.*—We may here again, for the sake of completeness, mention the progressive, sloughing, purulent, and fibrinous (diphtheritic) diffuse inflammations of cellular tissues, which have already been referred to. Putrid discharges which form in recent wounds, and which may quickly infiltrate into the meshes of the connective tissue, give rise on the second, third, or fourth day to those forms of inflammation of cellular tissue, which are characterised by an especially rapid and diffuse extension, and by products which tend to rapidly decompose, even while they are still in the tissues; if the patient should live through such a phlegmonous attack the process will terminate with necrosis of the suppurating subcutaneous cellular tissue. The same applies to the fibrinous (diphtheritic) phlegmons. Both processes are generally accompanied by very severe general symptoms. In already existing suppurations, phlegmonous inflammation may at any time spread around a wound as the result of mechanical irritation, foreign bodies, great congestion of wounds, the retention and decomposition of pus in diverticula, and from an infection with phlegmonous products from elsewhere, so long as the wound remains open.

2. *Hospital gangrene; ulcerative (phagedænic) wound-diphtheria pourriture des hôpitaux.*—I will first describe the disease, and then make a few remarks on its etiology. At certain times (seasons) it is noticeable, especially in hospitals, that wounds, not only recent ones, but also such as are nicely granulating and undergoing cicatrisation, begin to sicken in a peculiar manner, and that, too, without any apparent cause. The granulating surface becomes more or less converted into a yellowish slimy pulp, which can be partially washed off the surface, but which remains attached in its deeper layers. This condition spreads over the granulating surface, and it may also attack the bordering parts, which up to this time were quite healthy; the skin becomes reddened, and then covered with the same yellowish pulp; the surface of the wound may quite double itself in extent in about three days; the spreading into the deeper parts is in this so-called *pulpy form* of hospital gangrene comparatively trifling; at all events, the fascia and muscles offer considerable resistance. In other cases a recent

wound, or even a granulating surface, may rapidly assume a crater-like appearance, excrete a sero-purulent fluid, after the removal of which the deeper tissue lies exposed; the surrounding skin is slightly reddened. The progress of this molecular disintegration with sloughing generally takes place within certain well-defined areas, in consequence of which the wound is generally of a horse-shoe or trefoil shape. The ulcerative form of hospital gangrene spreads more rapidly than the pulpy variety, and tends also more decidedly to penetrate deeply. Although the two forms may occur quite independently, yet they may be seen in combination. I have seen the pulpy form more frequently than the ulcerative, but must confess that my experience of hospital gangrene rests on a comparatively small number of cases. It is not large wounds which are so especially subject to hospital gangrene; it may attack smaller and inconsiderable ones, such as leech-bites, and scarifications after cupping, and the sore produced by a blister even may become gangrenous; this disease never attacks an uninjured part.

The resemblance of wounds, attacked by hospital gangrene to diphtheritic disease of mucous membranes, has frequently been referred to by many authors. But after seeing a wound infected by the diphtheria of a mucous membrane I become more and more convinced that diphtheria and hospital gangrene differentiate themselves into two separate diseases both as to etiology and symptoms, though both are characterised by the deposit of fibrinous infiltration on the affected tissues. A wound which is inoculated with diphtheria becomes covered with a thick fibrinous membrane, the whole wound is infiltrated, and the surrounding parts become erysipelatous; then a great part of the infiltrated connective tissue necroses, and either melts away gradually or falls out in large shreds. On the other hand, there is not the spreading, pulpy degeneration of the cutaneous edges of the wound, with the swelling and tendency to bleed, which is so characteristic in hospital gangrene. After diphtheria, too, as is well known, peculiar paralyses are apt to occur. As a result of hospital gangrene these paralyses have not as yet been observed.

In hospital gangrene there is general constitutional disturbance, though the fever is not, as a rule, very high; there is, too, nearly always a certain amount of gastric trouble, the tongue gets coated, and there is also depression. To old and enfeebled persons hospital gangrene may become dangerous, especially if hæmorrhage have

occurred as the result of the invasion of small arteries. The large arterial trunks, as experience shows, often withstand hospital gangrene in a marvellous way. I once had a patient suffering from abscess in the inguinal glands, which had to be opened. Hospital gangrene of the pulpy form set in, and the skin, to the extent of a hand's breadth, was destroyed; the disease, too, spread deeply—so deeply that the femoral artery got laid bare for an inch and a half, and could be distinctly seen pulsating in the wound. I ordered a special attendant, who was never to leave the patient, and who was to be ready to compress the artery the moment any bleeding should take place, an accident which might happen at any time. The slough separated, and the wound began to granulate vigorously, the patient recovered completely, though slowly, without the slightest hæmorrhage. The erysipelatous redness which accompanies a diphtheritic phlegmon, as well as hospital gangrene, is generally sharply defined, and leads to desquamation, just as ordinary erysipelas does when it attacks a wound; but while the latter shows decided tendencies to migrate and to spread, the erysipelas of diphtheria and hospital gangrene for the most part remains stationary, or only spreads to a very limited extent. The general septicæmia is always more pronounced in diphtheria than in hospital gangrene.

The views held as to the cause of hospital gangrene are very various. This is no doubt in some measure due to the fact that many living surgeons have never had the good or bad fortune to see this disease. Thus, for instance, during the seven years I was at Zürich it so happened that no cases of either hospital gangrene or diphtheritic phlegmon occurred, although there was no lack of complicative wound-diseases. Surgeons who have never seen this disease, or only seen sporadic cases, believe that it is due to excessive carelessness, dirty dressings, and such-like causes, and think it in no ways differs from a superficial sore on the foot, which has become gangrenous as the result of dirt and neglect. Other surgeons believe that hospital gangrene is a disease which, as its name implies, is quite peculiar to certain hospitals, and that its spread is favoured by carelessness in the management and dressing of the cases. Another view is that this form of gangrene depends on epidemic miasmatic influences, and that the name *hospital* gangrene is thus far inappropriate that the disease may occur outside hospitals during the same epidemics. In the latter case the disease is propagated by infection, and I doubt not also that it may be

conveyed by forceps, charpie, sponges, &c., from a diseased to a healthy wound. Von Pitha and Fock are of opinion that hospital gangrene is an epidemic miasmatic disease. I had an opportunity of observing with Fock in the surgical clinic of Berlin an epidemic of this disease; it occurred during the same period in other Berlin hospitals, and also in private practice. It was proved conclusively that these latter patients had no sort of communication with the hospitals. The gangrene came on rather suddenly, and gradually disappeared after some few months, although the treatment of cases had in no way changed, and although no alterations were made within the hospitals. It is conceivable that hospital gangrene depends on the presence of minute specific organisms, which are only very seldom developed, and that they fall upon the surface of wounds, and there, acting as a ferment, give rise to decomposition. On this account I mostly compare this disease with blue suppuration, which, though it does not give rise to any serious consequences, is, according to Lücke's observations, like blue milk, caused by minute organisms, and is inoculable on other wounds. The requirements for the growth of these minute and undoubtedly vegetable bodies are probably more particularly favoured by certain atmospheric conditions, and in this manner an epidemic outbreak of the disease is brought about. It is certain that in the discharge from a gangrenous sore a large quantity of micrococci and streptococci is found just as constantly as in the secretion of ordinary diphtheritic sores. It is not possible to say certainly whether they are contained in the tissues previously to their decomposition, or whether they vegetate, and, so to speak, reduce it to a pulp; nor is it possible to say whether these micrococci are of a specific variety. It is certain, however, that the transference of hospital gangrene, either as pulp or pus, to healthy wounds, usually (according to Fischer, always) reproduces the disease, and this is a very important fact in practice. According to my own most recent researches in the Vienna General Hospital, I am persuaded that this disease depends on specific causes, and that it is altogether independent of pyæmia, septicæmia, erysipelas, and lymphangitis, though it may be followed by any of these diseases as complications.

The treatment must consist, first and foremost, in the strictest isolation of the patients, for whom there must be special attendants, special dressings, and instruments. If these measures do not

entirely prevent the spread of the disease (which can be carried from a diseased to a healthy wound by means of the atmosphere), they will nevertheless do much to hinder its spread. During some epidemics in military hospitals it has been necessary to completely evacuate the buildings. The local treatment must consist in the application of strong chlorine water, or spirits of camphor, or turpentine. Sometimes painting every two hours with the tincture of iodine has been found to answer remarkably well; also the acetate of alumina has, in my hands, answered well. It must be freely applied to the dressings, so that the wound is well soaked with it. The solution must not be too strong, nor too long continued. If this treatment does not suffice, the wound must then be freely cauterised, so that the eschar remain attached for six or eight days, as in an healthy wound. I find that the most effectual treatment is to apply fuming nitric or carbolic acid to the wound, and for a little distance beyond, and this must be repeated until the eschar firmly adheres. Perhaps it is safest to thoroughly clean the wound with lint or a pledget of wool, and so remove all slough, and then to apply the acid when it ceases bleeding. This operation must, of course, be done under anæsthesia, in order to do it thoroughly. The disease is by these means generally arrested. The general treatment must be strengthening and even stimulating. The pyrexia of hospital gangrene is due to absorption of decomposing matters, and differs then in no way from other forms of putrescent fevers.

There are two places in the body, in wounds of which the above-described pulpy phagedænic gangrene most frequently occurs, and that without any infection from without, viz. in wounds of the mouth, and in wounds of the urinary bladder. I mention this here because these diseases are without any doubt related to phagedænic diphtheria, although the description of them properly belongs to the domain of special surgery, on account of their localisation to special parts of the body. After extirpation of large portions of the tongue, and after resections of the lower jaw, I have seen, on various occasions, very rapid sloughing of the cellular tissue which had previously been indurated and extensively infiltrated. In such cases we have undoubtedly to do with a combination of diphtheritic phlegmon and phagedænic ulceration. Most of these cases terminate fatally by sepsis; a few get well, after the whole of the infiltrated tissue has become necrotic, and has freely suppurated away. Although the mucus and saliva with which these wounds so freely come in

contact do not possess, as such, any phlogogenous or infectious qualities, yet they may get mixed with the septic elements which collect in the mouth, and on the gums, and between the teeth; for it is difficult to cleanse the mouth on account of the sores; and on this account, indeed, it is frequently neglected. Thus the ferment mixes with the mucosity and saliva of the mouth, and on this account the term muco-salivary diphtheritis seems indicated. This disease generally comes on within the first five days; it seldom developes later. It is only fresh wounds which are attacked by the ferment-contagium in question; when good granulations have once formed diphtheritis never comes on, unless in consequence of an external infection, or from some mechanical injury to the granulations covering a wound. The general condition of the patient in this disease may be very serious; indeed, the patients are liable to sudden collapse, all the more dangerous in consequence of the debilitating influences which in many cases have preceded the operation, and reduced him considerably.

After lithotomy, urethrotomy, operations for perineal fistula, and ectopion vesicæ, pulpy destruction of the edges of the wound with a fibrinous deposit on the mucous membranes of the bladder, or vagina, as the case may be, is not uncommon, especially when the urine is alkaline; and as this disease is undoubtedly connected with decomposition of the urine, it is called urinary diphtheria. This particular form of diphtheritis is the mildest of any of the varieties we have alluded to, in thus far that it shows but little tendency to spread, and it may run its course without giving rise to any constitutional disturbance, provided always that the wound be kept thoroughly clean. It does happen, however, in certain cases, that the mucous membranes for some distance perish, and that the retro-peritoneal cellular tissue becomes involved in a purulo-sloughing phlegmon. The retro-peritonitis then sets up peritonitis, and this always terminates fatally. Diphtheritic inflammation about the vagina may lead to a superficial suppuration of the internal surface of the uterus; and this may spread up the tubes, and so reach the peritoneum. This variety is also generally fatal. In there latter cases, as also in the too frequent cases after parturition, and those rare cases after operation for vesico-vaginal fistula, severe constitutional disturbance is early manifest.

In the secretions from muco-salivary diphtheritis, and in urinary diphtheritis, micrococci and streptococci are constantly found.

They are also found pretty constantly in any lingual mucus, and in the fur on the tongue, and in ammoniacal urine; but they appear to develope further and with great rapidity in the discharges above referred to. The contagium peculiar to this discharge cannot at present be distinguished from the micrococci, and it may be assumed, therefore, that the latter contain, either within or around themselves, the contagious zymoid element. The proof that any micrococcus, wherever produced, may give rise to the process, is still wanting, though many observations plead in favour of the view, that these vegetable organisms take up certain contagious elements with an especial facility, and thus become the carriers both of contagion and of ferment. If inoculations be made with fluids which contain micrococci, for instance, on the cornea of rabbits, the cocci grow—as the interesting experiments of Nassiloff, Eberth, Leber, Stromeyer, Dolschenkow, Orth, Frisch and others conclusively show, to a certain extent, and then act (if not charged with any particularly noxious substances) simply, and for the most part mechanically, by forcing apart the layers of the cornea; the little colonies of cocci then gradually become surrounded by pus, which is discharged, carrying with it the cocci. In other cases (if the inoculated material contain any deleterious qualities) the whole cornea may become gangrenous within twenty-four hours, although the proliferation of the cocci is not half so extensive as in the former case. Then comes another series of cases in which the proliferation of the cocci produces no change in the cornea whatever, but disappears without leaving any traces behind it. After inoculations on the cornea of dogs this is indeed generally the rule.

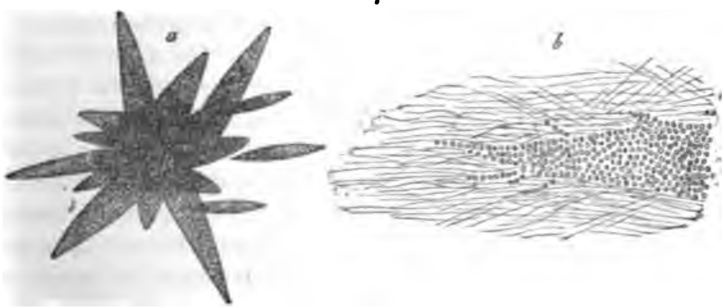
From what has been said it is evident that the kind and the intensity of the inflammation depend not on the proliferation of the fungus as such, but rather on the nature of the contagion which it brings with it, or gives rise to.

I thought it desirable to enter into these matters, in order that you might at least have some data to go on concerning this interesting and much-discussed subject. I can confidently recommend to your attention and study the excellent monograph, by C. Heine, on hospital gangrene.

3. *Traumatic erysipelas,* as already mentioned, belongs to the acute exanthemata; it is characterised by slight swelling, a rosy tint of the skin, which is painful also, and it is generally accompanied by fever. It stands in a peculiar relation to the other exan-

themata; in the first place, because it so frequently accompanies wounds, although it may appear quite spontaneously; on the other hand, it is propagated by a contagium which is considerably

FIG. 72.



(a) Appearance of the fungus on the cornea of rabbits. Proliferation of the cocci between the layers of the cornea after inoculation. Low power.
(b) A point of the same fungus under a higher power. × 600. After Frisch.

less infectious than that of measles or scarlet fever; and, lastly, one attack of the disease does not protect from a second; on the contrary, the patient is perhaps more liable to a second attack. As I scarcely ought to take for granted that you are already learned in skin diseases, I must briefly go through the symptoms of this disease.

The onset of the disease varies, for the pyrexia may precede the efflorescence, or the pyrexia may be synchronous with it. Let us suppose, for instance, that you have a patient with a small suppurating wound on the scalp, which is doing well, and beginning to granulate, and you find him one morning with a high temperature, and you hear, perhaps, that he has had a severe rigor. You examine the patient carefully all over, and fail to find anything beyond some slight gastric trouble, indicated by a coated tongue, a nasty taste in the mouth, a tendency to vomit, and loss of appetite. Such a condition of things is common to the early stage of many different diseases, so that you cannot possibly arrive at any diagnosis. Irrespective of the possibility of an accidental complication of some internal organ, one thinks of phlegmon, of lymphangitis, and of erysipelas. Probably twenty-four hours later we shall find the wound dry, secreting but a little serous fluid, the surrounding parts for some distance swollen, reddened and painful, and the granula-

tions also possibly œdematous and covered with a membranous deposit; the colour of the skin will be rosy red, and the redness will be everywhere sharply defined; the pyrexia continues. The diagnosis of erysipelas is now certain, and we feel pleased that, though not free from danger, the disease is one of the less dangerous ones which complicate the healing of wounds. In a second series of cases the erysipelas appears simultaneously with the pyrexia. We may hesitate for a short time whether we have to do with a lymphangitis, with an inflammation of subcutaneous tissue, or with an attack of erysipelas. The course of the disease will, however, soon determine this: the extent to which the erysipelatous inflammation of the skin spreads on the first day rarely remains the same, but increases little by little; the circular, patchy raised margin of inflamed skin is always well defined, and can be accurately mapped out as it passes first in one direction and then in another. The redness, in fact, spreads much in the same way as fluids do when dropped on to blotting paper. The disease may thus spread further and further, from the head to the neck, thence over the front aspect of the trunk, or over the back, or towards either arm, and lastly it may even involve one or both of the lower extremities. Pfleger has proved that the mode of spreading of erysipelas migrans is almost always the same, and that it most probably depends on certain lymph streams which are disposed in a certain manner in the meshes of the cutis. As long as the erysipelas spreads in this manner the fever generally remains high, and old and weakly persons are often and easily exhausted by it. Erysipelas generally lasts from two to ten days; fourteen days is an unusual period. The very longest case that I have seen lasted thirty-two days, and terminated favorably. You will also observe in those cases of erysipelas ambulans, or serpens, that the highest degree of inflammation of the skin only remains at a given point for a certain time, and then, as the erysipelas spreads, another part becomes the seat of the acme of the inflammatory process.

After the disease has lasted about three days in one place the redness disappears, the epidermis peels off, partly as a branny powder and partly in scales of various sizes. In many cases the epidermis is raised into blisters filled with a serous fluid at the very commencement of the disease, so called erysipelas bullosum. It must not be thought that this is a separate variety of the disease; it is but an expression of a more rapid exudation. In erysipelas

of the face it is not at all uncommon for blebs to arise, while in other parts of the body the erysipelas will have the usual appearance. If the disease attack the hairy scalp the whole of the hair may fall off, but it grows again very quickly. According to my own observation erysipelas starts most frequently in the lower extremities, then in the face, then the upper extremities, chest, back, head, neck, and abdomen. The scale of frequency probably depends on the comparative liability to injury of the various parts of the body given.

Erysipelas may supervene in other exanthemata or other internal diseases; as, for instance, pleurisy in erysipelas of the head, or meningitis even. On the whole, however, complications of this sort are rare, and then are mostly due to the spread of the inflammation to the deeper parts.

As regards the course of erysipelas, it may be said generally to run a favorable course. Of 137 cases (without complications) which I had under care in Zürich 10 died. Children, old people, and such patients who had got debilitated through other diseases are the least favorable subjects; and, according to my experience, they die of sheer exhaustion, on account of the persistent fever. On *post-mortem* examination there is no special change in any given organ which could be pronounced as the cause of death. Cloudy swelling and granular degeneration of the liver and kidney epithelium, softening of the spleen, are the appearances which are found, and which are common to all severe blood diseases. It is not quite clear in what the process of erysipelas consists, for neither the cause of its onset nor the mode of its spread is quite clearly understood. Dilatation of the vessels of the cutis, serous exudation into the tissue itself, over rapid development of the cells of the rete Malpighii, and cellular infiltration between the bundles of the cutis, can be anatomically demonstrated. The disease does not affect the subcutaneous connective tissue very much. In some places, however, it swells up to an enormous extent, as in the eyelid or scrotum, by becoming greatly infiltrated with serum. The œdema in most cases subsides again, without any further consequences. In a few rare cases, however, this œdema reaches such a degree that in consequence of the tension of the vessels the circulation of the blood ceases in these places, and in individual parts the erysipelas, for instance, may partially or altogether become gangrenous. If the entire upper or lower eyelid should be destroyed in this way a very

serious disfigurement would result. Fortunately, as a rule, only small portions are destroyed, and the skin of the eyelids, of the upper especially, is so ample that the defect can be easily remedied. In a few cases, at the termination of the erysipelatous process, a thickening of the subcutaneous tissue will persist in places, at which, occasionally, fluctuations may be detected, and from which pus may be evacuated.

There are many views as to the cause of erysipelas. The erysipelas of the head, which appears to come on spontaneously, without any wound, is most frequently attributed to cold. Some old people are said to get the disease every spring or autumn. Physical influences are said to be a cause; especially fright, chiefly in women at the menstrual periods, is said to produce erysipelas. Disturbance of the digestive function is also mentioned as a cause. I am very sceptical as to all these views, which unfortunately do not appear to rest on very accurate observations, but rather on tradition. I am very doubtful whether erysipelas ever develops except from a wound, or from some pre-existing cause of inflammation.

From what I have seen of traumatic erysipelas in hospitals I have come to the following conclusions. The local manifestation of the erysipelas I consider as an inflammation of the cutis, in which the specific poison is carried by the lymph vessels, through which the disease is thus further and further spread. The manner in which the inflammatory redness spreads, and is nevertheless sharply defined, renders it certain that its advance corresponds with the extent of certain lymph-vascular districts. By a careful observation it may be seen that very frequently, close on the margin of the red patches, there are at first circumscribed reddened, round spots, which merge into the already existing diseased parts, and which correspond obviously with the distribution of the branches of the lymph vessel. The same appearance is remarked on artificially injecting the skin from a small artery. At first the injection material runs in patches, and only becomes confluent after continued pressure. Veins and arteries have only spare communications with each other on the skin surface, but the lymph vessels, on the contrary, possess manifold intercommunications on the surface, but a less number of larger vessels in the subcutaneous tissue. The exciting poison spreads easily along the superficial lymph vessels in the cutis, like fluid in blotting paper, and

it may also spread more deeply through the subcutaneous lymph vessels, where it afterwards gives rise to both local inflammation and to mischief in the nearest lymph glands, as evidenced by streaky redness of the skin and tenderness and swelling of the corresponding lymph glands. When I speak of a septic or phlogistic poison as the cause of erysipelas, I only refer to the variety which we call traumatic erysipelas, because I believe, as the result of my own personal observations, to have demonstrated that the poison is generated in putrefying blood or certain other products of inflammation which are shut up in the cavity of a wound, or that the poison is directly brought from an infected wound to one which is otherwise healthy. As to the nature of this poison I can make the following remarks. It is probably a dried material in the form of dust, which can infect a wound at any stage; it clings especially to sponges and various surgical dressings. I have frequently observed that patients who on the same morning, one after another, and in the same operating theatre, and under the same conditions, have undergone surgical operations, have one and all developed erysipelas, the freshly made wounds have shortly become erysipelatous, without any retention of secretions, although the patients have been taken to different parts of the hospital widely separated from each other. In this manner erysipelas becomes endemic in a hospital; the infective material can attach itself to the clothes of the surgeons, or to instruments, and to the beds, and be conveyed in this manner; and it may attach itself also to the walls of the wards. The more carefully I think over my erysipelas cases in Zürich, and here in Vienna also, the more clearly do I see that they occurred in groups, a mode of onset which is entirely independent of any infective potentialities outside the hospital.

I have proved by my statistics, extending over two years, and also by communications from medical men of the canton of Zürich, that erysipelas had not occurred epidemically either in the town or country during this time; and further, like other exanthemata, it was especially frequent during the spring and autumn.

It is obvious, therefore, that epidemics of erysipelas in the hospital must depend on conditions (circumstances) which are to be found within the hospital itself, and which I have already referred to. The question here occurs, whether the poison which

produces the erysipelas is always the same; that is, whether the poison is a specific one. It is difficult to decide such a point. In favour of such a view, there is the fact that the kind of inflammation which is produced is always the same, though it may differ both in intensity and extent. On the other hand, it is to be remembered that the erysipelas may be produced by various products of decomposition, by miasmata, and very probably also by many other animal poisons. It is easily conceivable, however, that in all and each of these various poisons there is some specific matter which of all varieties of inflammation can only produce an erysipelatous one, or a material, materials, or fungous elements which find a soil peculiarly favorable for their growth within the lymph vessels of the cutis. It is too generally conceded that these materials develop and propagate under similar conditions more certainly and more freely at one season than at another.

The belief has frequently been expressed, and especially in recent times has been defended by Orth, that erysipelas is produced by the growth of micrococci on the surface of wounds, whence they are carried into the skin. Although the propagation and multiplication of the erysipelatous contagium resembles very closely the multiplication and propagation of a ferment, it has yet to be proved that the micrococci are the carriers of this ferment, still more, that they are the only ones. I cannot support such a view at all, though I have many times found cocci and streptococci in the serum of the vesicles of erysipelas; but, again, I have found them in other vesicles, in gangrene, in sudamina, in smallpox, &c., &c., and it is far from proved that these diseases depend on the presence of micrococci. It is very doubtful whether the inflammation produced on the skin of rabbits by inoculating them with erysipelas serum was identical with erysipelas on the human subject. The latest work on erysipelas by Lukomsky demonstrates very prettily the close connection between the growth of micrococci and erysipelas, and according to some observations made in my wards by Ehrlich, I can substantiate the truth of these experiments; but however interesting they may be, they fail to settle conclusively the difficult question of the etiology of erysipelas.

The disease always begins with rapidly rising temperature, the pyrexia continuing as long as the cellular inflammation goes on; sometimes it is continued, at other times remittent, and it terminates either in crisis or lysis. I have very little experience of

so-called spontaneous erysipelas of the head and face; but I believe that it is in the highest degree probable that it almost always proceeds from some slight wound (a scratch on the scalp or face), or from some inflammatory accident (nasal catarrh or angina), and that it is due to a septic cause.

The treatment of erysipelas is essentially expectant. We may act prophylactically by most carefully cleansing the wound, and removing anything which can favour its production, especially when there are other cases of the kind in the hospital; we must be especially careful not to allow too many patients in the same ward, and we ought further to see that our wards are allowed to remain empty for a while, for ventilation and cleansing purposes, so as to anticipate and prevent the development of a more intense erysipelas contagium.

As regards local treatment, a variety of measures has been adopted in order to stay the spread of the erysipelatous inflammation, and to bring it to an early standstill. For this purpose the margins have been painted with a solution of nitrate of silver, or with a strong solution of iodine. According to my own experience, this is of no avail, so that latterly I have quite left off the practice. Neither have I seen any advantage from the use of tar, which has been vaunted, and which was freely used in my wards for a time. The older surgeons believed that the inflammation was forcibly driven away by the use of cold, but that this favoured the production of inflammation of internal organs. Although we cannot consider this proved, there are nevertheless circumstances which seem to render the use of cold very inconvenient. We have already mentioned that, in cases with great œdema, gangrene may occasionally occur, and this would certainly be favoured by the application of cold; furthermore, the application of ice bags to large areas, such as the back or the face, is very inconvenient, and finally it is of little or no use, as the erysipelas runs its natural course, for in this disease especially the local manifestation and the general infection go hand in hand as in other inflammatory affections. The symptoms which the patient complains of in the affected parts are an uncomfortable tension, slight burning, great tenderness and susceptibility to the air and to changes of temperature. It is convenient, therefore, to cover up the diseased parts so as to protect them from the air. This can be accomplished in various ways. The simplest way, and the one I generally adopt, is to

smear the skin over with oil, and then to cover it with cotton wool; the patients generally like this method. Others bestrew the affected parts with flour, or hair powder, or sift some finely divided camphor on the wool, in the hope of producing some local effect. If vesicles arise, they may be pricked with a needle, and the epidermis pressed down and allowed to dry.

If there be any tendency to gangrene anywhere, a dressing with chlorine water, or some other antiseptic, may be used. Abscesses which form in the subcutaneous tissue after an attack of erysipelas must be opened early and treated in the usual manner.

Internally nothing is required beyond some cooling drinks. If there be any appearance of failing strength, or if the disease seem likely to last for a long time, you must then prescribe tonics and stimulants; such as a few grains of camphor daily, quinine and a little wine.

Any complications arising in internal organs are to be treated, of course, lege artis; in cases of meningitis, do not hesitate to apply ice-bags to the head, even if the scalp is the seat of erysipelatous inflammation.

4. *Inflammation of lymph-vessels; lymphangoitis, or lymphangitis;* more properly, inflammation of lymph trunks, is of frequent occurence in the extremities as the result of various causes, of which I will now speak. The appearances, in the arm for instance, are the following—The hand gets a small scratch or wound; the arm becomes painful, especially on making any movements; then the glands in the axilla become swollen and tender, even when lightly touched. On carefully examining the arm red streaks will be found especially on its flexure surface, which run lengthwise up the arm from the wound to the inflamed glands. These reddened patches are very sensitive. At the same time there is pyrexia, a coated tongue, malaise, loss of appetite, and general depression. This may terminate in one of two ways; with proper care and treatment the inflammation generally subsides, the red streaks disappear, as also the swelling and tenderness of the axillary glands. In other cases suppuration occurs; the skin of the arm gets reddened by degrees, and for a considerable extent becomes œdematous; then the swelling of the axillary glands increases, the temperature rises, and rigors sometimes occur. In the course of a few days fluctuation becomes evident, generally in the axilla, sometimes, however, in the arm; then, either spontaneously or after an incision, a

quantity of pus is discharged from a more or less circumscribed abscess cavity. Now the pyrexia declines, the pain and swelling also, and the patient soon recovers from a disease, which, while it lasts, is both painful and debilitating. But the termination is not always so favourable, for even pyæmia may, and does occasionally, occur, as the result of lymphangitis after poisoned wounds; the pyæmia is usually of the subacute form, of which I shall have to speak hereafter. I remember seeing a patient, who was also suffering from some chronic kidney affection, with a lymphangitis of the leg; the inguinal glands were affected; shortly afterwards the superjacent skin became enormously œdematous, and finally gangrenous. This mode of termination is exceedingly rare, although the pus in cases of abscess in the lymphatic glands, especially after *post-mortem* sepsis, very often has a foul, ichorous quality. Acute inflammation of the lymphatic glands (lymphadenitis) with termination in suppuration may occur as an idiopathic disease. We conclude, at all events, that such is the case, from cases where we are unable to find any streaks of connection between the inflamed glands and a wound or other inflammatory focus. It is, nevertheless, doubtful whether this view is always correct, for the superficial lymph trunks are only perceptible when inflamed, while the deeper ones cannot be appreciated either by the eye or the finger. We are able, therefore, to recognise only a superficial lymphangitis. It is one of the peculiarities of this disease rarely to spread beyond either the axilla or the inguinal region when it affects the extremities. I have once seen a pleurisy supervene on the same side in the case of lymphangitis of the arm and adenitis of the axilla, which most probably resulted from infection conveyed along the lymph vessels.

As to the pathological anatomy of lymphangitis of the subcutaneous connective tissue we know very little, scarcely more than we can see with the naked eye in our patients; for so long as the disease confines itself to the lymphatics it is scarcely ever fatal, and because the disease can only be imperfectly produced by experiments on animals. At any rate, the surrounding connective tissue is seriously implicated, and the capillaries dilated and filled with blood. Whether the lymph trunks become plugged later on with coagulated lymph, or whether from the very commencement clots form in the lymph (which is less coagulable than blood) that set up irritation in the lymphatic vessel walls, we are unable to decide. If we apply

our observations to the lymphangitis uterina, such as frequently occurs in puerperal fever, to inflammation of the skin, we may venture to expect to find pure pus in the dilated lymph vessels in certain stages of the disease: the parts immediately surrounding the peri-uterine lymphatics are infiltrated with cloudy swelling; the plastic infiltration of cellular tissue passes on to a purulent infiltration, or even to the formation of an abscess, in which the thin-walled lymphatic vessels terminate; the more circumscribed the lymphatic plexus the more difficult is it to distinguish lymphangitis from an inflammation of the cellular tissue. Thanks to the drawings of Cruveilhier ('Atlas,' livr. 13, Pls. ii and iii), we can get a very excellent idea of lymphangitis puerperalis, and can then apply this to lymphangitis of other parts. The red streaks which we see in the skin can only be produced by dilatation of the blood-vessels around the lymphatics, and not by the passage of blood into these vessels. Thus, then, we only see in our patients the appearances of a peri-lymphangitis, resulting from contact with the septic materials contained within the lymph vessels. We can appreciate the changes in the lymph glands, however, a little more exactly; the blood-vessels circulate freely within them, and thus the whole tissue becomes saturated with serum; large masses of cells fill out the alveoli, and this probably then interferes with the circulation of the lymph within the glands, then entirely stops it, and thus, to some extent, in consequence of some arrested lymph circulation, the diseased process comes to an end, or at any rate is limited. Lymphangitis can, indeed, complicate any wound or inflammatory centre; but my own opinion is, that it is the result of septic materials circulating through the lymphatic vessels. The poison may be of the most varied kinds—decomposing products in a wound, putrid matters of all kinds (especially *post-mortem* poison), which in consequence of over-irritation form in wounds. We have already said that a simple scratch from a hob-nail may become a diffuse inflammatory centre, in which a phlogistic poison, may be, and often is, formed, which leads to lymphangitis; and so it is with inflammatory foci from other causes: in consequence of some increased irritation a material is formed which reacts in a very irritating manner in the efferent lymph vessel and its immediate neighbourhood. Further, an encapsuled poison in an inflammatory focus may, by increased blood tension, get into a lymphatic vessel, and thence into the blood, although without such cause it might have remained quiet and have been gradually

excreted or eliminated by suppuration. I will quote the following case as an instance in point. One of my colleagues had a small inflamed sore on his finger, caused by a *post-mortem* examination; it was quite local, and he paid but little attention to it. While on an Alpine tour he got considerably overheated, and one evening a lymphangitis of the corresponding arm set in, with great pyrexia. In consequence of his over-exertion, and of the correspondingly increased heart's action, you see that the poison which was lying quietly in a small, circumscribed inflammatory focus, generalized itself and got into the blood. Why, in different cases at one time we have a diffuse phlegmonous inflammation, at another erysipelas, and at a third lymphangitis may depend either on purely local circumstances, or on the quality of the septic material. It is impossible to say anything definite on this point. According to the well-known observations concerning the emigration of cells from the vessels, it is conceivable that pus-cells, which are generated in the wound, and thence carried into the lymphatic circulation, wander out through the walls of the lymph vessels, and as conveyers of irritating material of some kind set up perilymphatic inflammation, while the more quickly circulating toxic fluid in the centre of the lymph stream gets into the blood and sets up fever, before the local inflammatory process is at all considerably extended.

The treatment of lymphangitis in recent cases must always be directed in such a manner as to bring about resolution, and to prevent suppuration. The patient must keep his affected limb as quiet as possible; and if there be any gastric troubles an active emetic will be of service. The disease, indeed, but seldom recedes after the aperient and diaphoretic action of the emetic has set in. Of the local means, the inunction of the whole limb with mercurial ointment is very efficacious. In addition, the part must be kept warm, so that a slightly elevated temperature may be kept up. For this purpose either cotton wool may be used or not, or fomentations may be tried. If, in spite of this treatment, the inflammation continues to increase, and if redness and swelling set in, then suppuration will most probably occur at some spot or other. Under such circumstances hot fomentations are indicated. This kind of diffuse inflammation does not by any means confine itself to the lymphatic vessels, but the whole of the subcutaneous cellular tissue becomes more or less involved. As soon as fluctuation becomes manifest at any place, an incision must be made in order to let out the pus. If

the healing process is slow, a daily warm bath will prove of great service. Baths are especially indicated in those cases where there is a tendency to relapses. A septic material encapsuled in the lymph glands can, by once again getting into the circulation, set up peri-adenitis. In this way the frequent relapses, seen after an apparent and long period of latency in this disease, can best be explained.

LECTURE XXV.

5. *Phlebitis, Thrombosis, Embolism.—Causes of Thrombosis in the Veins.—Various Metamorphoses of the Thrombus.—Embolism; Red Infarctus, Embolic Metastatic Abcesses. Treatment.*

5. *Phlebitis. Thrombosis. Embolism. Embolic metastatic abscesses.* —In addition to the forms of inflammation already described, another process is frequently observed, commencing in a wound or inflammation-nest, at first local, but then extending in a peculiar manner to several organs, namely, phlebitis and thrombosis. In persons dying from this disease we find pus and crumbling suppurative or ichorous coagula in the thickened or partly suppurating veins in the neighbourhood of the affected parts. In addition to these formations, abscesses frequently occur in the lungs, more rarely in the liver, spleen, and kidneys. That these metastatic abscesses are connected with the pus in the veins has already been shown by Cruveilhier; the nature of this connection has, however, not been explained until much later.

What I communicate to you to-day on this subject is the result of a long series of investigations and experiments, for which we are indebted to Virchow, and which have so often been repeated and confirmed by many others that no doubt of their correctness can be entertained. I have myself devoted much attention to this subject, and will point out, as occasion serves, where I have arrived at results differing from those of Virchow. It would lead me too far if I attempted to develop for you historically the course of those comprehensive works of Virchow, and to give them again here in a summary form; I must leave it to your own diligence to study them for yourselves, and content myself with presenting to you the chief results in a short review.

The first question of great importance is: *in what relation does the coagulation of the blood stand to the inflammation of the vessels?* We know from the investigation of the formation of the thrombus after the tying of arteries, and from the study of the healing process in injured walls of veins, that coagula of blood form at once in the injured vessel before any inflammation of the walls of the vessel can be observed. The coagulum which forms in veins after injuries to them, and represents the thrombus, is indeed in most cases very short; but it can easily be imagined that it may become greatly enlarged by continuous deposits of fresh fibrinous matter. You know physiologically that the fibrin can be separated from the blood by stirring and beating it until coagulation takes place. During the movement of the blood, coagulating fibrin attaches itself, like crystals, to rough surfaces, and you can easily convince yourselves experimentally that such a surface, *e.g.* a cotton thread, introduced into a vein of a living animal, soon becomes covered with fibrin. Roughnesses of various kinds in the vessels may, therefore, occasion coagulations of greater or less extent. Such roughnesses may no doubt arise from disease of the walls of the veins on their inner surface, and coagula be formed in this manner. Small abscesses in the walls of the veins may cause projections into their area, and it was even assumed formerly that fibrinous coagulation occurred on the inner surface of an inflamed vein as on an inflamed pleura. Whether this happens can scarcely be determined; what was formerly considered as such has already been shown to be a peripheric layer of the blood coagulum deprived of its colour. Such abscesses in the walls of the vessels are, at all events, extemely rare, and can, therefore, but very seldom give rise to coagulation. *It much more frequently happens that the coagulum formed in the vessels after the injury, forms, under certain conditions not to be ascertained exactly, the starting-point for further coagulations, and eventually for inflammation of the walls of the vessel.* After injuries, a second point comes under consideration, by means of which coagula may be formed in the vessels, viz. retardation of the circulation resulting from frictional obstruction, *e.g.* contraction of the vessels themselves. This mode of formation of coagula may be designated as *thrombosis from compression.* This also is not directly dependent upon inflammation of the wall of the vessel, but may arise from inflammation of the tissues surrounding the veins. It may happen, namely, in a case of very violent inflammation, that a tissue, espe-

cially if subjected to a certain amount of pressure by a fascia, may become so much swollen, partly from serous, partly from plastic infiltration, that the vessels thereby become compressed, and stasis and coagulation produced. *This thrombosis from compression in very acute inflammation, and especially in acute incidental inflammation of the cellular tissue in the neighbourhood of wounds, is much more frequent than primary traumatic thrombosis; it is also the most dangerous form of thrombosis, because most frequently followed by puriform breaking up of the thrombi.* In addition to the mechanical compression, which favours coagulation, *the inflammation of every tissue includes another factor which is followed by the same consequences, namely, the changes which the inner coat of the vessels, and especially of the veins, undergoes therein.* If we do not know the positive chemical conditions which are required for the coagulation of the blood in the vessels, we know from the valuable investigations by Brücke that the normal, living inner coat of the vessels possesses the especial quality of keeping the blood fluid, and that coagulation sets in whenever the inner coat loses its normal character. But it loses its normal character in the veins as well as in the walls of the capillary vessels through the inflammatory process itself, as is seen from the most recent investigations into inflammation. These investigations show, it is true, that the inflammatory change in the walls of the vessel, as such, does not cause at first either complete stasis or thrombosis. It is, however, not improbable that the formation of the latter is at least facilitated by that change in the walls of the vessels. The most recent phases of the theory of inflammation would thus justify, for many cases at least, the earlier view that inflammation of the wall of a vein may cause thrombosis (even if no abscess form in it); at all events, further investigations into this point are very desirable. Chemical observations favour the view that such a course of things exists, for it is well established that phlebitis and thrombosis are often preceded by inflammation in the neighbourhood of the veins, a *periphlebitis* analogous to perilymphangoitis. Also, with rapid dilatation of a vessel, the course of the blood is considerably retarded in accordance with physical laws, and coagulations take place at the diseased dilated point, as we shall see later on in the case of aneurisms and varices. This is termed *thrombosis from dilatation.*

Further, the course of the blood may be greatly retarded by deficient and weak contraction of the heart and arteries. Since this

occurs chiefly in persons much weakened by age or by long and exhausting diseases, this form of thrombosis has been termed *atrophic* (marantisch). It appears to be quite independent of inflammation of veins, and occurs most frequently in parts at a great distance from the heart.

You must bear in mind in connection with all these forms of thrombosis, that they at first occupy a small district only, and gradually increase in size by the constant deposit of fresh fibrin. *Why* the traumatic thrombosis extends so abnormally in many cases of injuries to veins is only intelligible in those cases in which extensive crushing has produced extensive lacerations of the veins and considerable consequent obstructions of the circulation. But, for those cases in which widely-extending thrombi follow a simple punctured or incised wound of a vein, *e. g.* bloodletting, the cause is extremely difficult to explain, and not possible for all cases. Traumatic thrombosis and thrombosis from compression and their consequences must occupy our special attention, while atrophic thrombosis and thrombosis from dilatation are more rarely met with in surgical cases. It has been asserted that thrombosis in the veins, followed by suppuration, is much more common in hospitals than in private practice, and the tendency to coagulations has been attributed to the hospital air and to the miasms suspended therein. That the hospital miasma (a something indefinable in itself, but undoubtedly of a very complex nature), as such, directly occasions coagulations of the blood, is an assertion which can neither be proved nor refuted. In my opinion, an indirect connection only is probable; by means of toxico-miasmatic infection of a wound, whether brought about by infection from instruments, dressings, or otherwise, acute suppurative inflammations are produced in the neighbourhood of the wound, sometimes in the form of ordinary inflammation of the cellular tissue, sometimes in that of diffused lymphangoitis, diphtheritis, and similar processes. These inflammations first produce the thrombosis in the veins, just as in very acute phlegmonous inflammations originating outside the hospital; the influence of the miasmatic poisoning upon the production of thrombosis in the veins is, therefore, not a direct one, but only an indirect one, resulting from the infection of the wounds and consequent inflammation.

Our next task will be to inquire *what becomes of the blood coagulated in the vessels, and in what relation the wall of the vessel stands to it.* We are acquainted, as yet, with but one metamorphosis of

the thrombus following injuries to the arteries and veins, namely, its organisation to form connective tissue. This is extremely rare in extensive thrombosis in the veins, and then naturally leads to complete obliteration of those vessels. Let us confine ourselves to the consideration of a perfectly simple case, thrombosis after bloodletting. After this operation, in the median vein, for instance, an acute, more or less extensive inflammation of the cellular tissue sets in, generally resulting from the use of unclean instruments or bandages. With this inflammation of the cellular tissue, which spreads periphlebitically, a coagulation of blood takes place as well in the injured vein as in the cephalic and basilic veins, downwards as far as the wrist, and upwards as far as the axilla. In consequence of the interruption to the circulation thus caused, the œdema of the whole arm increases considerably; and when this swelling has subsided, we can feel the subcutaneous veins quite plainly as hard cords beneath the skin. The after course of things may vary; first of all, a termination in dispersion may take place, and with early treatment usually does so; the patient must keep his bed, since he generally has some fever, the arm must be kept absolutely at rest, and should be enveloped in a compress thickly covered with grey ointment. Under this treatment the swelling of the arm will often decrease and the fever cease. The hard, venous cords can still be plainly felt for some time, but become softer in the course of six or eight days, and eventually cease to be distinguishable. We seldom have an opportunity of examining cases of this kind anatomically in the early stages. We cannot, therefore, ascertain to what extent the walls of the veins are already diseased during the process of coagulation, or whether they are so at all; but it appears, at least, from the symptoms and examination of patients, that the fibrin coagulated in the vessels gradually becomes redissolved and mixes again with the blood without injury to the latter, like blood which had spread as a diffused extravasation in the tissues and afterwards become absorbed.

The second mode of termination which occurs in inflammation of the arm after bloodletting, and is combined with thrombosis, is the formation of abscesses. The first symptoms are as described above, but then there appears, either at the bend of the elbow or on the fore or upper arm, a more circumscribed inflammatory swelling, which enlarges steadily, and finally presents distinct fluctuation. On making an incision, pus comes away from a larger or smaller cavity, the swelling in the arm decreases in size, the abscess heals

from within, and the cure may become complete. The anatomical investigation of these cases shows that suppurative inflammation has been developed, commencing in the cellular tissue surrounding the veins. We become convinced, further, that the coats of the thrombosed veins are much thickened, which is regarded by some as a consequence, by others as the cause of the thrombosis. I will add here at once that the diagnosis of venous thrombosis on the ground that the vein feels like a hard cord is not always a safe one, since the inflammatory process in the cellular tissue sometimes takes place, as already stated, exactly and at first only around and along the veins, and thus a tube-like thickening of the sheaths of the vessels occurs, which may easily be confounded with thrombosis, but by no means necessarily leads to it. This confounding of a periphlebitic induration of the cellular tissue with thrombosis has happened to me twice in connection with the saphena vein, and I look upon it as impossible to form an exact diagnosis in all cases. That such a periphlebitis, in which the coats of the veins no doubt participate to some extent, may exist without thrombosis, is proved more than sufficiently by the fact that the latter is not always necessarily the cause of inflammation of the veins, as was asserted formerly.

If circumscribed suppuration set in around a portion of vein thrombosed over a considerable area, the vein itself and the portion of it surrounded by the abcess then suppurate also; the thrombus becomes organised above and below the abscess, merges in the wall of the abscess which has become converted into granulation tissue, and the venous stumps become obliterated. This is the reason why hæmorrhage scarcely ever occurs from these abscesses in phlebitis. A further metamorphosis which the thrombus may undergo is that of crumbling decay. The softening of the coagulum then generally commences at the point at which the thrombosis began, *i.e.* in the oldest part of the coagulum. The fibrin breaks up into a pulpy mass, which sometimes assumes rather a yellowish, sometimes rather a brownish colour and greasy consistence. This decay eventually spreads further and further; the inner coat of the veins also does not remain unaffected, but becomes thickened and wrinkled. The thrombus thus becomes converted into pus, which mixes with the detritus of the fibrin, while the walls of the veins and surrounding cellular tissue become much thickened. It also sometimes happens that small abscesses form in the walls of the veins, but this is rare. The pus found in the vein under these circumstances has not been

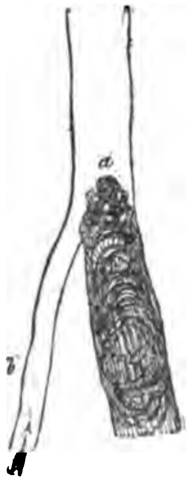
absorbed from the wound, as was believed formerly, but has been formed in the vein itself from the coagulum. The purulent-looking fluid is frequently nothing else but fluid detritus of fibrin (in purely atrophic thrombosis without inflammation), while, in many cases, healthy, thick pus, with well-developed pus corpuscles, may be observed in these veins. If the wound is in an ichorous state, the fibrinous detritus may also assume an ichorous character in the vein, while probably, in consequence of the capillarity of the thrombus, ichorous fluid is taken up out of the wound, and the decaying fibrin thereby infected. It is also possible that this capillarity of the thrombus may cause the decomposing wound-secretion to affect the blood. It is evident that there can be no question of an extensive mechanical passage of pus or any other secretion from the wound through the vein into the current of the blood, since the opening in the vessel is blocked up by the thrombus. Should rapid decay of the thrombus in the vein, *as far as the peripheric and proximal end,* set in, which seldom occurs to this extent, venous hæmorrhage must first take place, and when this ceases, a fresh thrombus must be formed, so that there also a passage of the pus of the wound into the vein, and of the pus of the vein into the blood, could not take place. Moreover, the pus formed and collected in the vein is, so long as the proximal end of the thrombus remains unaffected, also so isolated that it cannot mix with the blood; this can only occur if the proximal end of the thrombus decays altogether; but this seldom happens, because, in most cases, fresh deposits of fibrin occur constantly while the decay goes on from the oldest portion of the thrombus. You will understand, therefore, that the entrance of pus into the injured veins cannot, in general, easily occur, but that very special conditions, to be mentioned presently, must exist to render this possible. I must now break the course of my description for a short time to mention that Virchow does not distinctly recognise the conversion of the thrombus into pus. I have no doubt on this point; if the blood corpuscles in the thrombus possess, in general, the capability of increasing and of becoming converted into tissue, as still appears probable to me, there is no reason why we should deny to them the power of forming pus in the thrombus any more than to the white corpuscles of the circulating blood escaping from the vessels, for the coagulation of the blood is by no means so firm as to prevent completely the movements of the corpuscles. That the thrombus may become converted

into true pus by the breaking up of the white corpuscles of the blood I regard as not yet disproved; *that this for the most part encysted pus never or only very rarely* enters the circulation, and consequently does not generally stand in any direct relation to pyæmia, has been mentioned already. If I try to sum up my experiences of thromboses in the veins, and of the fate of the thrombi, they would appear to show *that the majority of such thromboses are the result of very acute inflammation of the cellular tissue (especially beneath fasciæ, tight skin, and in bone), and that the coagulum undergoes the same metamorphosis as the inflamed tissue.* If the inflammation lead quickly to formation of tissue, the thrombi in the vessels also become organised and form connective tissue; if the inflammation run into suppuration or ichorous decomposition, the thrombi also suppurate or become ichorous, and break up into a crumbling mass. This is now so much less difficult to understand since we know from the investigations of Recklinghausen and Bubnoff that the corpuscles may pass from the tissue through the walls of the veins into the thrombi. The walls of the veins themselves undergo the same process under these circumstances as the thrombus and the surrounding tissue; they become plastically infiltrated and thickened, or they suppurate.

A thrombosis with phlebitis may run its course as a purely local process, as not unfrequently happens with phlebitis after blood-letting and in many other cases. A further danger may arise from thromboses with crumbling, suppurative, or ichorous decay of the coagulum. The proximal end of the thrombus, as we have already pointed out when speaking of thrombus in the arteries, usually extends as far as the next entering branch of the vessel, with a slightly pointed conical end; the latter also projects even a little beyond the opening of the former (Fig. 73A), and if the coagulum is no longer quite firm, a piece of it may be torn off by the passing blood and reach the circulation. This passes into larger and larger veins and at last into the right side of the heart and thence into the pulmonary artery, in the branches of which it eventually usually stops at a point of bifurcation, being too large to proceed further. The respective branch of the pulmonary artery is now closed by the fibrinous coagulum as if by a cork, a so-called *embolus,* and the first consequence will be the cutting off of the supply of blood to the portion of the lung supplied by that arterial branch. This local want of blood (ischæmia, Virchow) does not, however, gene-

rally continue long, but blood enters into the empty arterial branches and that for the most part from a retrograde movement of the venous blood, as Cohnheim has demonstrated. Under certain

FIG. 73.



a. Proximal end of a venous thrombus projecting into one of the larger branches; *b*, a lateral branch not thrombosed; the blood flowing through the latter may detach the point of the thrombus (*a*) and carry it into the circulation. Diagrammatic drawing.

circumstances, the district deprived of blood becomes overfilled and coagulation ensues. In such cases, lacerations of vessels and hæmorrhages also occur, and since the arteries of the lung, spleen, and kidneys, go on dividing into smaller and smaller branches, and their district towards the periphery thus becomes constantly larger, and resembles a wedge projecting with its point into the respective organ, so must the district in which the coagulation here described has occurred also have a wedge-like form. The term *"red or hæmorrhagic, wedge-shaped infarctus"* has been applied in pathological anatomy to these coagulations resulting from embolism. Frequently as these cases of wedge-shaped infarctus occur, their origin is not an absolutely necessary consequence of embolism, for if the embolism does not affect precisely an arterial terminal branch, and the collateral arterial circulation is powerful enough to drive the blood into the artery beyond the embolus, as happens in otherwise healthy individuals and in animals, as well as in many cases in

which the emboli produce but little mechanical or chemical irritation of the tissues, no infarctus is formed, nor does any considerable interruption of the circulation ensue, and we have to deal only with the local processes set up around the embolus as a foreign body situated in the branch of the artery. These local processes depend upon the character of the embolus; *if the latter consist entirely of coagulated fibrin, a slight thickening of the wall of the vessel takes place at the point at which the embolus is situated, and the latter may, if surrounded by fresh coagula, become organised and form connective tissue, or it may be absorbed. If the embolus consist of a fibrinous coagulum impregnated with pus or ichor, it excites, not only in the wall of the vessel, but also in its neighbourhood, a suppurative or ichorous inflammation.* The metamorphosis of the red infarctus is, therefore, dependent partly upon its size, partly upon the force of the circulation still existing here and there in it, but partly also, as observed above, upon the chemical character of the embolus. If the latter be quite indifferent, and the infarctus very small, or if it be still fed by some vessels not thrombosed, the coagulum forming the infarctus may become absorbed, or organised so as to form connective tissue and a cicatrix. If the embolus be indifferent, while the coagulation in the whole infarctus is complete, tissue and coagulum both gradually break up into a yellow, granular, dry mass, which is surrounded by a capsule and may become chalky; this is the *yellow, dry infarctus.* If the embolus be impregnated with ichor or pus, it excites ichorous or suppurative inflammation in the whole neighbourhood; the infarctus then also undergoes ichorous or suppurative decay and *suppurative or ichorous abscesses form.* As we are speaking here chiefly of the lung, I may at once mention that these abscesses, which are situated for the most part at the periphery, often cause pleurisy, that they are most frequently multiple in both lungs and may even lead to suppuration of the pulmonary pleura at the point corresponding to the abscess and occasionally to pneumothorax.

You cannot easily imagine, gentlemen, how much labour was required to prove so clearly this connection of thromboses in the veins with the abscesses in the lungs as to enable me to present it to you here as a simple fact. You will read with admiration the classical works on this subject by Virchow, Panum, O. Weber, Cohnheim and others; it would lead me too far to enter more into detail about them here; we claim the right at present to pluck only

the ripest fruits from this luxuriant forest of writings. We have now clear notions concerning embolic infarctus and abscesses in the lungs, but how does it stand with the infaretus and abscesses which are met with, although much more rarely, under similar conditions, in the spleen, the liver, and the muscles? Are these also always dependent upon emboli? A few years ago, we should have been unable to answer this question with certainty; we can now do so in the affirmative. It has been proved experimentally, chiefly by O. Weber, that certain kinds of emboli, especially flakes of pus, can pass without obstruction through the capillary vessels of the lungs into the left side of the heart and thence into the general circulation, where they may stick fast in the spleen, liver, kidneys, or elsewhere, and cause abscesses. This serves to explain those rare cases in which, with thrombosis in the veins, abscesses do not exist in the lungs, but are met with in other organs. If, together with abscesses in the lungs, embolic infarctus or abscesses exist in the domain of the general circulation, the further explanation is admissible that venous thromboses with suppurative or ichorous decay have been formed from the abscesses in the lungs, pieces of which reach the left side of the heart and pass onwards.

The embolic origin of *metastatic abscesses* has been so clearly demonstrated that we may now draw safe conclusions from the existence of such abscesses concerning venous thromboses with suppurative or ichorous softening. As regards the proof of such a connection in an individual case, it may be very easy in many cases, but also frequently very difficult: very easy when we have to do with thromboses of *large* venous trunks and emboli in large *branches of the pulmonary artery* attainable with the scissors; very difficult when it is only a question of coagulations *in small venous nets* (*e. g.* in phlegmonous inflammations and in gangrenous bedsores), and of embolisms in capillary districts of the *lung, spleen, kidneys, liver, muscles, &c.*, and yet these latter cases are extremely frequent. That embolisms occur in capillary vessels is proved clearly in individual cases in especially favourable objects (*e. g.* in the capillaries of the brain), and there is no doubt that small veins become thrombosed in all cases of suppurative inflammation. Clearly to show this anatomically in every case is very difficult, and often impossible. From what symptoms we infer whether a coagulum is old or fresh is taught in the lectures upon pathological anatomy; you will there also be told how you can distinguish small lobular infiltrations in

the lungs, such as occur especially in suppurative bronchitis, from metastatic abscesses. I will now only add that it sometimes happens that a venous thrombus remains firmly organised at the point at which it enters into the wound, while its upper part suppurates and breaks up completely, and eventually becomes carried entirely into the circulation through collateral branches in which the blood circulates. That is the only case in which pus in the veins reaches the general circulation when there has been no hæmorrhage. We recognise this process after death from the fact that we find in the thickened vein, rough on its inner surface from layers of thombus still adhering to it, fluid blood or perfectly fresh coagula formed after death. If the respective portion of the vein becomes involved on the formation of a periphlebitic abscess, the proof of a previously existing thrombus undergoing suppurative decay can no longer be furnished with absolute certainty. We are speaking here only of metastatic *circumscribed inflammations*, of infarctus and abscesses; these alone stand in relation to venous thrombosis and embolism. As regards diffused metastatic inflammations, another explanation must be sought for, of which more when speaking of septicæmia and pyæmia. Neither will we dwell longer here upon the febrile conditions in phlebitis, or in the establishment of metastatic processes. Since phlebitis with its consequences is, for the most part, only an incident in already existing acute inflammations, it is difficult to judge how far the former, in itself, gives rise to fever; metastatic abscesses will, doubtless, like all other inflammatory nests, be followed by fever; from a simple thrombosis of a vessel, as such, fever can scarcely be expected to set in. We can certainly cause fever in dogs by the production of numerous small embolic nests in the lungs by means of the injection of starch or finely powdered coal into the jugular vein, as has been shown by Bergmann, Stricker, and Albert, but the same thing cannot be effected with certainty with embolisms in other parts of the vascular system, and may perhaps be dependent upon other conditions not yet exactly ascertained.

As regards the *treatment* of phlebitis and thrombosis, it is the same as that of lymphangoitis and other similar acute inflammatory processes. Cautious inunction with mercurial ointment, or, if detachment of the coagulum is to be feared, covering the inflamed part with a compress smeared therewith, bladders filled with ice, and absolute rest of the affected part are indicated. Of the diagnosis

and treatment of metastatic abscesses at the bedside we will speak later on in connection with pyæmia. If phlebitis and thrombosis terminate in local suppuration, we must open the abscesses as soon as we have been able to diagnose them.

LECTURE XXVI.

II. GENERAL INCIDENTAL DISEASES WHICH MAY SUPERVENE UPON WOUNDS AND INFLAMMATION-NESTS. 1. WOUND- AND INFLAMMATION-FEVER; 2. SEPTIC FEVER AND SEPTICÆMIA; 3. SUPPURATIVE FEVER AND PYÆMIA.

II. *General Incidental Diseases which may supervene upon Wounds and other Inflammation Nests.*

The local, incidental wound-diseases hitherto described are always connected with disease of the whole system; this general affection is predominantly of a febrile character, although not always so. Fever is such a complicated assemblage of phenomena that it may assume a very different appearance on the supervention of one symptom or another. By general agreement the existence of fever is now recognised only when there is an increase in the temperature of the blood and the intensity of the febrile process is estimated by the height of that temperature. I am not inclined to differ much from this proposition, because, by refusing to accept it, we should lose a uniform conception of that which we term "fever," and throw back the theory of fever into its former chaos. But I must here already point out to you that there exist many and even very dangerous general diseased conditions in the wounded and in individuals with other inflammation-nests, in which no increase whatever in the temperature of the blood is demonstrable. The latter is, therefore, *only conditionally* a measure of the degree of danger in which the patient finds himself. Besides the temperature of the blood we have, in fever, the following chief symptoms: acceleration of the pulse and respiration, want of appetite, frequently combined with nausea, a feeling of weakness, profuse perspiration, not unfrequently violent jactitation of certain groups of muscles (in rigors), more or less mental excitement, and inertness of the sensorium.

Fever is a general diseased condition which may arise from a great number of causes. According to the quality and the quantity

of the matters which find their way into the blood from the inflammation-nests (either from wounds or also directly through the lungs and from the alimentary canal), which we may designate as fever-exciting (pyrogenic) poisons, certain symptoms present themselves more prominently. Thus we have fever with very high temperatures on the subsidence of all other symptoms, fever with predominating inertness of the sensorium, with but slight increase of temperature, fever with predominance of violent shiverings, fever with predominant disturbance of the functions of the stomach, fever with a predominanant feeling of exhaustion, &c. Why should we not admit a form of fever in which all the other symptoms except an increase of temperature are present? Precisely this symptom might, on some occasion, from some cause or other, be masked or prevented from being recognisable. But, as already said, we will content ourselves with the definition of fever at present in use, and admit its existence only when increased temperature of the blood is demonstrable; but we must add that serious, general, incidental wound- and inflammation-affections exist, which run their course without fever.

There is another uniform element in the affections here in question of which we must not lose sight, namely, *that they all arise from the absorption of matters which have their origin in the wound or its neighbourhood, or*—what is much the same thing—*in an inflammation-nest.* This absorption takes place through the lymphatic vessels and walls of the veins.

I imagine that the absorbed matters, partly in a state of solution, partly so finely granular as to pass through filtering paper, advance rapidly with the current of the blood in the centre of the veins, but only very slowly on their inner surface and in the lymphatic vessels, since the blood itself advances very slowly along the walls of the vessels. In consequence of this, the poisonous matters penetrate gradually into the walls, and through them into the surrounding tissues, where they produce the periphlebitic and perilymphangoitic inflammations already mentioned, while the matters carried forward with the central stream in the vessels rapidly become mixed with the blood.

Herein we agree with the present views as regards wound-fever, inflammation-fever, septicæmia, and pyæmia, less perhaps when it is also a question of tetanus, delirium tremens, delirium nervosum, and acute mania. There are, however, important grounds for regarding the latter diseases also as being of humoral origin, for which reason I shall not further subdivide the diseases here in question.

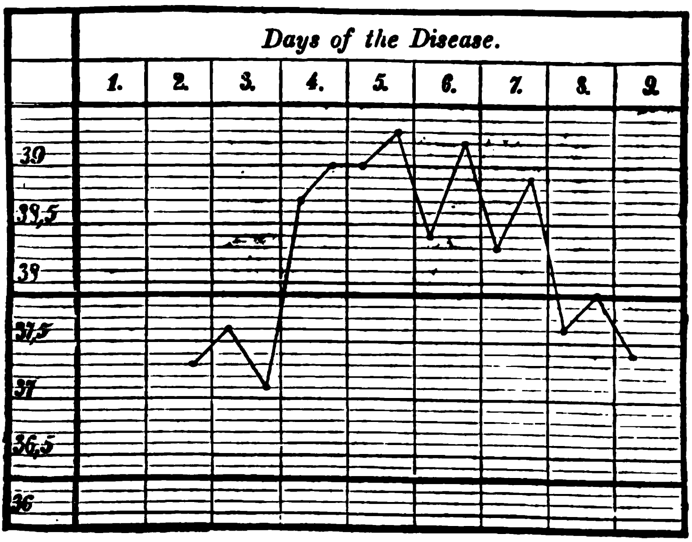
1. *Wound- and Inflammation-Fever.*

I have already explained that the fever which occurs in the wounded arises partly from the taking up of matters which originate in the decaying tissues on the wound-surfaces, partly from the taking up of matters which are formed in the tissues during the traumatic or incidental processes of inflammation. With this assumption, which we sought briefly to justify on a former occasion, it will depend partly upon the local conditions for absorption, partly upon the quality and quantity of the respective pyrogenic matters absorbed, how great the degree of poisoning will be. There are cases in which such a rapid closure of the vessels laid open by the injury, and such a rapid circumscription of the whole traumatic inflammation-nest occur that, in the first instance, no general infection or fever whatever is set up, nor yet perhaps at all later on. These cases are rare with extensive injuries, and may be regarded as ideal normal cases, in which the plastic infiltration at the edges of the wound leads rapidly and over the whole extent of the latter to a new formation of firm organised tissue fitting closely into the edges of the wound, either with direct conversion into cicatricial tissue or with previous formation of granulations. If we accept these cases as normal types, every wound-fever is a pathological incident. We must admit this in theory, but in the generality of cases with wounds of any considerable extent, fever supervenes sooner or later, and on that account it appeared necessary, when describing the general condition of the wounded, to speak of wound-fever. Much remains, however, to be added to what has already been said which would have been difficult to you to understand at an earlier period. Let us first of all speak *of the time at which the wound-fever usually appears and of the course which it runs.* In many cases, especially in those in which the injury occurred in tissues previously healthy, the fever does not commence until the second day, rises rapidly, continues with morning remissions for some days at a certain height, and then gradually (seldom within twenty-four hours) ceases entirely. According to my very numerous observations, the wound-fever begins far most frequently within the first forty-eight hours after the injury. It is customary to represent these febrile movements graphically in the manner shown in Fig. 74.

The curve shows that after a primary amputation of the arm,

rendered necessary by an injury, where it happened that no observation was taken on the first day, the fever did not set in until the third day, then continued until the seventh day; this patient then remained free from fever from the eighth day, while in other cases secondary fevers not unfreqently occur after amputations.. Such a course of wound-fever is pretty common. I explain this to myself in the following manner:—Immediately after the injury, the tissue of the edges of the wound was closed by plastic infiltration; on the third day this began to suppurate and become mixed with shreds of decayed tissue on the surface of the wound, and there was thus set up a moderately extensive inflammation of the amputation-stump, with absorption of pus and other products of decay and inflammation; this absorption went on until it was stopped on mechanical grounds (diminished pressure, thickening and partial closure of the vessels, &c). In other cases, the fever commences

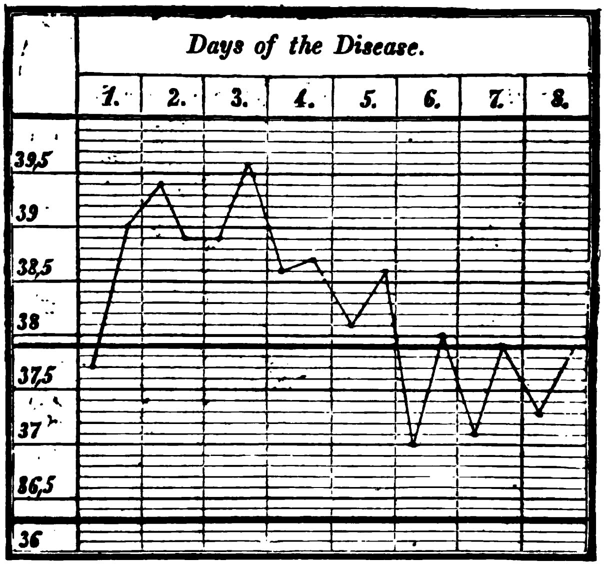
FIG. 74.



Fever curve after amputation of the arm. Recovery. The ordinates of this and the following fever curves show the scale of the thermometer according to Celsius, every degree is divided into ten parts, the abscissæ signify the days of the disease; the curve is drawn according to the observations taken daily, morning and evening; the two broad lines signify the maximum of the highest and minimum of the lowest normal temperature in healthy individuals.

on the very day of the injury; we meet with this not unfrequently, on the one hand, when blood has been shut in between the edges of wounds sewn together and rapidly become decomposed, and also when operations have been performed in tissues which have undergone inflammatory infiltration. The following case may serve as an instance of the second (fig. 75):

FIG. 75.



Fever curve after resection of a carious wrist-joint, with great infiltration of the soft parts. Recovery.

In chronic-inflammatory infiltrated portions of tissue the finest lymphatic capillaries are perhaps contracted and partly closed, and on that account already have not for a long time carried off properly the serum from the tissue, but the middle-sized lymphatic and venous trunks, which have long been subjected to increased pressure in chronic inflammation, are undoubtedly dilated, and perhaps, on account of rigidity of the tissue, partially gaping, and thus, unless very rapidly closed by plastic infiltration, take up from the very commencement a large quantity of the wound-secretions. This explanation of mine of the earlier or later setting in of the wound-fever is purely hypothetical; but it is founded upon

numerous observations, and has developed itself in my mind from them. It might also be assumed that the matters taken up into the blood act very slowly in one case, very quickly in another, but this is far from probable. So long as the earlier view still prevailed that fever always resulted from an irritation of the nerves, it was necessary to bear in mind that such irritation, and consequently its febrile action, might be set up at very different periods. I have abandoned this view without, however, underestimating the important part which the nervous system plays in the production and phenomena of fever.

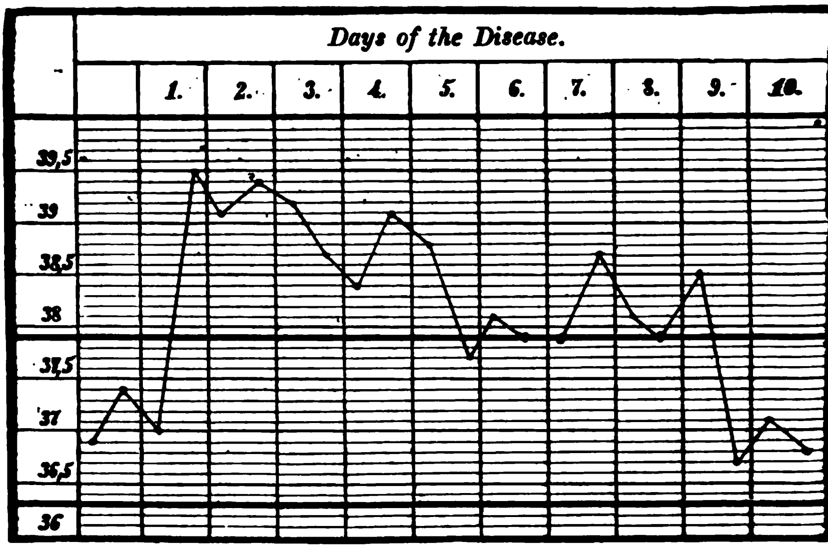
The duration of wound-fever is generally about seven days; at least it is seldom longer without visible local complication.

If an incidental inflammation set in around the wound, whether of the cellular tissue, the lymphatics, or the veins, the fever (which appears as *inflammatory secondary fever* either in direct connection with the wound-fever, or after several or even many fever-free days), occurs simultaneously with this inflammation, or apparently precedes it. I say apparently, because we may often have overlooked the first beginnings of the local process in such cases, either because they perhaps offered no striking phenomena, or because the poisonous matter infected the mass of the blood more rapidly than the surrounding tissues. The course of such secondary fevers depends entirely upon the course of the local inflammatory processes; with the commencement and spread of the latter the temperature rises rapidly, frequently with initial rigors. The longer such secondary fevers continue, the longer, that is, the poisoning goes on, so much more dangerous does the state of the patient become; rapid emaciation, profuse perspirations, sleeplessness, and constant want of appetite are the evil symptoms. Very pronounced erysipelas or violent inflammation of the trunks of the lymphatic vessels and of the lymphatic glands are the comparatively most favorable forms of incidental inflammations, because they generally lead in a shorter or longer time to a distinct, for the most part favorable termination, and thus, to a certain extent, possess something of a typical character, although the duration of an erysipelas may vary from three to thirty days or more, and cause extreme prostration. The fever curve shows at first a rapid rise, then remains about a certain height, generally with morning remissions, not unfrequently followed by a rapid fall of the temperature; the same holds good for lymphangoitis. It rarely happens, fortu-

nately, that erysipelas or lymphangoitis spreads deeply into the subcutaneous cellular tissue, or beneath the fasciæ; the case would then enter into the series of the severe phlegmonous inflammations and lose entirely its partly typical character.

The fever in diffused, deeply extending inflammations of the cellular tissue, with or without venous thrombosis, does not always commence so suddenly, but has always from the first a very pronounced remitting character, and is, in its further course, like the

FIG. 76.



Fever curve in erysipelas traumaticum ambulans faciei, capitis, et colli commencing after the extirpation of cancer of the lip. Recovery.

local process, not to be calculated. The loss of strength, the emaciation, the irritability of the patients attain the highest degrees. An intermitting type of fever and metastatic inflammations, those chief symptoms of the malignant wound-fevers which we designate as "pyæmia," are, in such cases, always much to be feared.

In all these fevers, the quantity of urea is always greatly increased and generally exceeds the quantity of nitrogen contained in the food. At the same time, according to recent investigations, the weight of the body decreases considerably.

So long as the general symptoms, especially those connected with fever, do not go beyond what has been described above, and especially so long as death does not ensue, we usually content ourselves with the terms "wound fever, suppurative fever, secondary fever." But if other symptoms supervene, and death follows, two other terms for such very violent infections have come into general use, namely, "septicæmia" and "pyæmia." We will adhere to these accepted terms.

2. *Septic Fever—Septicæmia.*

By septicæmia is understood a, for the most part, acute general affection, which arises from the taking up of various kinds of putrid substances into the blood, and it is believed that these putrid substances so change the quality of the blood that it can no longer fulfil its physiological functions. We can cause this disease in animals by injecting ichor into the blood or subcutaneous cellular tissue, and experience has shown that larger animals especially (large dogs, horses) may, under certain conditions, survive the ichorous poisoning of the blood, although much weakened thereby. Special conditions are required for the taking up of ichorous substances into the blood of human beings; an absorption of such substances through the healthy skin and mucous membranes only takes place if the putrid substances themselves are, at the same time, destructive, corroding, or eventually attained an active penetrating power. Diseased portions of skin, or raw surfaces, on the contrary, take up such ichorous substances more easily, but again only under certain circumstances; these substances do not, for instance, easily penetrate well-organised, uninjured, granulating surfaces. We may cover a healthy granulating wound in a dog with lint dipped into the most offensive ichorous matter, and, unless the latter contain some corroding substances capable of destroying the granulations, the animal will not become diseased; the ichor is not absorbed. From this I conclude that the poison must be prevented in some way from penetrating into the blood-vessels situated upon the surface of the granulations. From these special conditions under which the infection by means of putrid substances usually takes place, it appears to be proved that the respective poison is unable to penetrate the mucous substance of the granulations, or that it is chiefly taken up by the lymphatic vessels, as I already mentioned formerly. If you further bear in mind that, in the case of contused wounds, decaying shreds

of firm connective tissue, especially of tendons and fasciæ, often remain for a long time lying upon the otherwise healthy granulating wound without any passage of septic poison from them through the superficial blood-vessels of the granulations into the blood, this observation serves to complete the experiment in dogs. I will not deny that perhaps, under certain dilated conditions of the walls of the blood-vessels, as well as by means of capillary attraction, infectious matters *may* also pass into the blood from thrombi in the vessels; neither will I deny that cells *may* take up molecular septic substances and carry them into blood-vessels; but I am inclined to regard this mode of infection as exceptional, especially if the infectious substances are not in a state of solution, but consist of minute granules, and are taken up, for instance, in the form of dust. It has been said in objection to this that undissolved granules in the blood could not act deleteriously, because only substances in solution are capable of poisoning the blood. That is quite correct; but we know that metallic mercury is also not soluble in the blood, and yet, in the form of grey ointment applied to the skin, may cause poisoning, *i.e.* profuse salivation. We do not know whether or how the quicksilver becomes dissolved in the tissues, but we see that it also acts when introduced undissolved into them. One remark more I must make, namely, that septic substances may undoubtedly be forced through granulating surfaces and the walls of abscesses into the tissues, the lymphatic vessels, and the veins. The conditions of pressure about wounds and in inflammation-nests and in the cavities of abscesses are of great importance clinically, and it is very desirable that they should be carefully watched and studied. This can be done better at the bedside, however, than here.

As regards the sound parts of the body which are exposed to the air, it has hitherto been proved for the lungs only that substances in the form of dust (coal) can pass into them, and thence into the bronchial glands, and presumably into the blood, while the absorption of undissolved substances into the chyle and blood-vessels of the intestines has not yet been observed nor found to be producible experimentally.

Many attempts have been made recently to ascertain what substance in the decomposed animal tissues is the actual poisonous principle, and for this purpose decomposing fluids have been treated chemically until a substance was at last obtained which produced, in

very minute quantity, the phenomena of septic intoxication. Thus Bergmann has obtained from decomposing beer-yeast a crystallising body of that kind, which he calls *sepsin*. To show that this body *only*, which Fischer could not obtain from decomposing serum or decomposing pus, is the poisonous principle, it would be necessary to prove the innocence of all other chemical bodies resulting from the process of decomposition. But this is not the case; sulphuretted hydrogen, sulphuret of ammonium, butyric acid, leucin, and many other substances resulting from the decomposition of organic matters act, when injected into the blood, more or less septically. It appears to me very probable that in decomposing fluids, according to their nature, the degree of their concentration, their temperature, &c., very many different poisonous bodies are formed, which I can further represent to myself as being in a state of constant change until they reach a final stage; whether such final stage is always the same is a question not yet solved. This is not the place for discussing these difficult questions at length; so far as my experiences, observations, and studies reach, I consider it at least in the highest degree probable that the septic matters are already formed in the inflamed and gangrenous tissues and pass into the blood as efficient poisons. This view stands in opposition to another, according to which the ferment, which only exercises its destructive fermenting or decomposition-exciting power after entering the blood, passes into that fluid from the tissues (originally from the air, O. Weber). According to this view, the septic matters absorbed are not, in themselves, poisonous, but first generate the poison in the blood itself from some of its constituent parts.

Quite recently this hypothesis has been more closely defined to the effect that the excitors of fermentation are cocci (monads, Hueter) or bacteria. I cannot agree with this view because I could not find micrococci either in the blood of living persons who died of septicæmia, or in the blood of such persons soon after death. I must further add that I was also unable to find, some time after the operation, cocci and bacteria in the blood of living animals into whose bodies I had injected fluids containing those organisms, and which died of the septic poisoning; neither could I find them in the blood of those animals some hours after death. It appeared from this as if cocci and bacteria not only do not increase in the living blood, but even soon die in it. According to these observations, we are not justified in assuming the existence of a hæmato-

zymotic process from organisms in septicæmia. Numerous works on these highly interesting questions have failed to furnish any decisive explanation. According to the investigations of A. Hiller and E. Anders, it appears certain that the much-discussed putrid poison is not only attached to the micro-organisms, but also exists in a state of solution and detached from them in the fluids acting as poisons, but yet not in every case. Especially remarkable appear to me A. Hiller's experiments, in which the blood of a rabbit killed by means of a putrid fluid free from bacteria proved, when injected into another rabbit, to be again fatally poisonous and so on, with increasing effect, many times over. In the present state of our knowledge this can only be explained by assuming the presence of a constantly renewed and increasing inanimate ferment. When Davaine first made known the same experiments with a fluid containing bacteria, followed by the same results, which were afterwards confirmed by Stricker, it was thought almost certain that such processes were only explainable by means of a *living* ferment; the necessity of accepting this view has now again become less pressing from the above-mentioned experiments of A. Hiller. At all events more attention must be paid in further experiments to separating, if possible, the "septogenic ferment" from the final product of the process of fermentation, the "septic poison." This will probably cost much labour; it is quite conceivable that the septogenic ferment, in itself, is a septic poison for many animals.

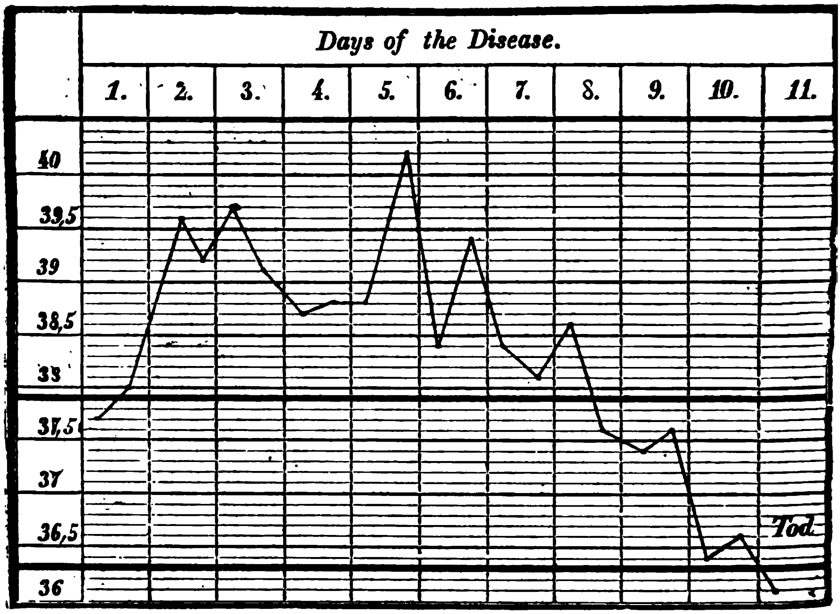
After these general remarks we will take into consideration those surgical cases which furnish opportunities for septic infection. First of all come the cases in which decomposition takes place in recent wounds; whether intense, unusual, local and general infection will occur, generally becomes evident during the first three days. If the local infection present itself in the form of moderate inflammation, which soon leads to healthy circumscribed suppuration, and the general infection be followed by slight fever only, the affection belongs to the class of simple wound-fevers; but if the local affection be very extensive, and phlegmonous inflammation with ichorous products ensue, while the general condition assumes a special character to be described further presently, we employ the term "septicæmia." In other cases there is a more extensive gangrenous nest resulting from a wound, or occurring spontaneously (*e.g.* gangrene from diseased arteries), from which the absorption of decomposing matters follows, and this happens more frequently and

intensely with moist than with dry gangrene. In a similar manner the condition for the absorption of putrid substances is furnished if, after the birth of the child, the placental surface of the uterus become gangrenous. Some of the cases of puerperal fever are cases of septicæmia.

It will be intelligible to you *that the conception of the disease "septicæmia" rests essentially upon an ætiological basis*, as, for instance, also the group of diseases "typhus," and that the milder septic wound-fever stands in the same relation to septicæmia as typhous febricula to typhus, and it has even been proposed to employ the term "septic febricula." But as typhus in its individual forms is characterised symptomatologically and pathologico-anatomically, so is this also the case with septicæmia, although its pathologico-anatomical characteristics are but slightly marked. But by what is the course of septicæmia characterised? When are we to speak of a severe case of wound-fever as "septicæmic?" Here the symptoms connected with the nervous system are to be especially noted; the patients are apathetic, sleepy, or even comatose; fearful excitement is less frequent; furious, maniacal delirium sometimes exists. The subjective feeling is good; the patients do not suffer much. The tongue is dry, frequently of a woody hardness, which gives to the speech of these patients something peculiarly laboured; the patients are thirsty, but seldom satisfy their thirst because they feel it but little on account of their general apathy. Frequently, but not always, profuse diarrhœa sets in, more rarely vomiting. At first there may be profuse sweating, later on the skin is dry and has a withered appearance. The urine is scanty, very concentrated, sometimes albuminous. As the disease advances, the patients pass their stools and urine under them. Gangrenous bedsores upon the sacrum form very early. The fever generally runs high at first; intercurrent rigors in the course of the disease *never* occur in a case of simple acute septicæmia; initial rigors are also a very rare symptom. In the further course of the disease the temperature falls to the normal point, seldom below it, and the patient generally dies in a state of the most complete collapse, with a thready, very rapid pulse. The condition of the pulse and tongue are more important for the prognosis of the septicæmic condition than the temperature. A contracted and rapid pulse and a dry tongue are unfavorable symptoms; normal temperature therewith has no prognostic value, while very high or very low temperatures

no doubt render the prognosis still less favorable. The agony often lasts more than twenty-four hours.

FIG. 77.



Fever curve in septicæmia after extirpation of an enormous lipoma between the muscles of the thigh. Death.

This is the regular course in cases of simple acute septicæmia occurring after a recent injury; the patient may, however, die in the first stage with a rising temperature. There are, further, cases in which the commencement of the fever is scarcely marked at all by a rise in the temperature, and, lastly, cases which run their course entirely without fever or with an abnormally low temperature. The latter occurs with a subacute course, especially in elderly subjects with spontaneous gangrene; but the other symptoms already mentioned are then, for the most part, all present. We see from this, as also especially from the above curve, *that the falling of the temperature is by no means, in itself, a sign of improvement,* but that also the other general symptoms—strength, state of sensorium, tongue, pulse—must be taken into account. The most serious cases of all are those in which, somewhere about the middle or towards the end of the second day, extreme *collapse with cyanosis* suddenly

sets in, which then generally causes death in a few hours. Such patients make exactly the same impression as cholera patients in the cold stage, except that in septicæmia vomiting and constant diarrhœa are seldom present. The patients appear as if suddenly poisoned, after having perhaps felt quite well during the first twenty-four hours after the operation. Just in these cases, which may also be combined with diphtheria, the secretion from the wound is by no means ill-smelling; the nose detects no signs of decomposition. It is not demonstrable whether the poisonous matter in these cases differs from the usual one, or whether the inflammatory change in the tissues here furnishes an especially large amount of poisonous products. The difference in the features of the disease in septicæmia is, according to what I have said above, pretty considerable; this proves nothing, however, against the assumption that the septic poison is always the same, for similar differences exist in the phenomena of cholera, gangrene of the spleen (Milzbrand), diphtheria, or the bites of snakes, in which diseases we do not on that account recognise different kinds, but only differences in the intensity and quantity of the poison absorbed, and differences in the power of resistance in individuals.

I hope that you will have formed for yourselves from what I have said a correct idea of septicæmia. The *prognosis* is, in strongly marked cases of the disease, extremely unfavorable; we will speak of the treatment at the end of this part.

We now come to the *post-mortem appearances*. We occasionally have some difficulty in finding again in the dead body the œdematous infiltration and the blueish and brownish discoloration of the skin which we observed in the neighbourhood of the wound during life. In other cases, which ran a longer course (six to eight days), we find the subcutaneous cellular tissue steeped in a bloody, serous fluid; in cases running a still longer course (a fortnight or more), there is generally very extensive suppuration of the cellular tissue, with more or less extensive gangrene of the skin. The internal organs often present no appearance of disease whatever. If constant diarrhœa existed during life, there is often swelling of the solitary and conglobate glands of the intestines. The spleen is frequently enlarged and softened, seldom normally large and firm; the liver is, for the most part, vascular, flabby, perhaps, also strikingly friable, but without any further change. The blood in the heart is frequently lumpy, partly coagulated, pitchy; in rare cases, firmly

coagulated, lardaceous; the lungs are in most cases normal. We frequently find slight diffused pleurisy on one or both sides, perhaps also traces of pericarditis; the kidneys are often swollen, and the serum scraped from their cut surfaces turbid. I will speak more fully of these diffused, metastatic inflammations not resulting from embolism when on the subject of pyæmia; this is not very important here, any more than the embolic infarctus and ichorous abscesses also observed exceptionally in septicæmia when the patients resisted the disease for a considerable time, and thromboses formed in the veins in the neighbourhood of the wound or gangrenous nest.

Since nothing peculiar has hitherto been discovered by chemical analysis of the blood after death in these cases, it must be admitted that the post-mortem appearances present but little that is characteristic of the disease. The features of the disease are essentially of an ætiologico-symptomatological character; if we had not seen the patient during life we should often seek in vain in the dead body for a palpable cause of death.

Many surgeons prefer saying that the person injured or operated upon succumbed to a severe typhous wound-fever, rather than employ the term "septicæmia." No etymological objection can be made to this, although I cannot regard it as practical; the word "typhous" is here applied in its older sense, as τυφος was used by Hippocrates for "heaviness, stolidity;" later on, the term "typhous fevers" was used quite generally to describe febrile states in which the patients are "stupid;" during the last twenty years only, distinctly characterised infectious diseases related to each other have been classed under "typhus." It is perhaps best to leave this as it now is, and not again to generalise the expression "typhous." Virchow, it is true, also uses the term "ichoræmia" in the sense in which I use septicæmia, but the attempt to reintroduce that expression does not appear likely to succeed.

3. *Suppurative fever, pyæmia.*

Pyæmia is a disease which we believe to arise from the taking up of pus or of the constituent parts of pus into the blood. It stands in the same relation to simple inflammatory fever and secondary fever as septicæmia does to simple, primary wound-fever, and is especially striking symptomatologically on account of the intermittent occurrence of accessions of fever, pathologico-anatomically on account of the very frequent occurrence of metastatic abscesses

and diffused metastatic inflammations. Synonymous terms for this disease are—metastatic purulent dyscrasia, purulent disease, purulent diathesis.

That you may be able to form for yourselves an approximate preliminary idea of this disease, I will describe to you a case of pyæmia. A patient is brought into the hospital with a compound fracture of the leg immediately above the ankle-joint, and an extensive contused wound. The injury was caused by the falling of a very heavy weight upon the part. You examine the wound, discover an oblique fracture of the tibia, but believe the injury to be of such a nature that recovery is possible. You therefore apply a dressing and the patient goes on at first very well, with but little fever, until about the third or fourth day. The wound now becomes more inflamed, and secretes comparatively little pus; the skin in the neighbourhood becomes œdematous, red, the patient has more fever, especially in the evening, the swelling around the wound increases and extends gradually. The whole leg is swelled and red, the ankle-joint very painful, and on making pressure upon the leg, thin, ill-smelling pus escapes with difficulty from the wound; the swelling is confined to the leg, the sensorium is not affected, and there is no sign of intense, acute septicæmia. The patient is extremely sensitive when the dressing is renewed and very low-spirited, a febris remittens continua, with rather high evening temperatures and a decidedly rapid pulse, has set in, the pulse is full and tense, the appetite is lost entirely, and the tongue is thickly coated. It is now about the twelfth day after the injury. A large quantity of pus flows from several parts of the wound, and somewhat above it distinct fluctuation can be felt. This suppurating cavity may indeed be emptied into the wound with difficulty by pressure, but the exit is much impeded, and it is best, therefore, to make an incision at that point. When this is done, a moderate quantity of pus comes away, soon after which the patient has violent rigors, then dry, burning heat, and eventually profuse perspiration. The appearance of the wound improves somewhat, but this does not last long; we soon observe in its neighbourhood, more posteriorly in the calf of the leg, a fresh suppurating cavity; rigors occur again, fresh counter-openings become necessary at various points, to provide a proper escape everywhere for the immense quantity of pus secreted. The left leg is the one injured; some morning the patient complains of violent pain in the right

knee, which is somewhat swollen and painful when moved. The patient passes sleepless nights, eats scarcely anything, but drinks a great deal, is much reduced and emaciated, especially in the face, the skin becomes slightly yellow, and the rigors recur. The patient now begins to complain of oppression at the chest and coughs a little, but expectorates only a small quantity of mucous sputa. On examining the chest you find, as yet, only a moderate amount of pleuritic exudation on one or both sides from which the patient does not suffer much; so much the more does he complain of the right knee, which is now much swollen and contains a large quantity of fluid. Since the patient sweats very much, the urine becomes very concentrated and sometimes contains albumen. At last bedsores form, but the patient does not complain much of them; he lies quietly now, partly comatose and muttering in a low voice. It is now about the twentieth day after the injury; the wound is dry, the patient looks very wretched; the face and neck are especially wasted, the skin of a deep yellow colour, the eyes dull, the tongue, protruded tremulously, is quite dry, the skin cool, the temperature low, and rising somewhat in the evening only, the pulse small and rapid, the respiration slow, and the breath has a peculiar, cadaverous odour. The patient becomes completely unconscious, and may, perhaps, continue in this state for twenty-four hours before death takes place. You now make the post-mortem, and find nothing pathological in the cranial cavity; the contents of the pericardium and heart are normal; in the right ventricle and auricle there is a firm, white, fibrinous coagulum; the cavities of both pleuræ are filled with a turbid serous fluid; the surface of the lungs is covered with a network of icteric layers of fibrine. You pull this off and find beneath it, in the substance of the lung, but especially at its surface, pretty firm nodules of the size of a bean or chestnut. These are found predominantly in the lower lobes, and show, in section, that they are for the most part *abscesses.* The somewhat thickened parenchyma of the lung forms the capsule of a cavity which is filled with pus and decayed lung tissue. Others of these nodules appear of a blood-red colour when cut through, their section-surface is somewhat granular, in their midst is found here and there pus in varying quantities, and it is evident that from them the abscesses proceed. You have here the *red infarctus* terminating in the formation of abscesses with which you are already familiar. Some of these abscesses lie so near the

surface that the pleura also becomes implicated, so that the pleurisy has set in secondarily. The liver is rather vascular and friable, but nothing further abnormal is observable in it. The spleen, which is somewhat enlarged, shows in section some firm, wedge-shaped nodules, with their apices inwards and their broad extremities directed towards the surface. They behave similarly to the red infarctus in the lungs, and are also partly in a state of suppuration at the centre. The whole intestinal canal, as well as the urinary and sexual organs, show nothing abnormal. On making an incision into the right knee, which was painful during life, a large quantity of flaky pus is poured out; the synovial membrane is swollen and partly injected, and the cartilages of the joint have lost something of their shining appearance. The examination of the wound shows nothing beyond what we had observed during life, namely, extensive suppuration of the deep and subcutaneous cellular tissue and pus in the ankle-joint. The walls of all these suppurating cavities consist, for the most part, of decaying tissue and true granulations have been developed at a few points only. The fracture is, however, more complicated than we had supposed, since a longitudinal fissure extends into the ankle-joint and at the posterior part of the tibia, where we could not make any examination during life, several detached pieces of bone are situated. In the veins of the leg we find here and there fibrinous plugs of some standing, perhaps also yellow puriform detritus, and at some points true pus.

You may now make some preliminary reflections upon this case, and represent to yourselves that you have observed a series of similar cases, so that it has become clear to you that it is not a question of an accidental combination of different diseases, but of a thoroughly connected entity. You have before you extensive, constantly increasing suppuration in one of the extremities and intense, continuous fever with more violent accessions. Upon this supervenes suppuration in a very distant joint, then circumscribed inflammations, with formation of abscesses, occur in the lungs and other organs. These multiple inflammation-nests keep up constant fever and, at the same time that the functions of the diseased organ become deranged, the organism succumbs under the signs of exhaustion. The peculiar and essential character of the disease consists, as you may easily observe, in the formation of numerous inflammation-nests after the primary suppuration has reached a certain point. You know the explanation of the formation of the

metastatic abscesses; they always result from thromboses and emboli in the veins, and require no further notice here. More difficult to explain are the *diffused metastatic inflammations* which occur as well in septicæmia as in pyæmia; they by no means always depend upon abscesses in the lungs, like the pleurisy in the case mentioned; we meet with diffused metastatic inflammations of the eye, the membranes of the brain, the joints, the subcutaneous cellular tissue, the periosteum, the liver, the spleen, the kidneys, the pleura, the pericardium, &c., which are independent of abscesses and, so far as we yet know, of embolisms. An exact explanation of the origin of these metastases is scarcely possible for all cases. If the metastatic nest stand in close connection with the original suppuration-nest, we may imagine the former to be produced by a continuation of the inflammation from the latter by means of the lymphatic vessels; in cases, for instance, in which, after amputation of the breast or exarticulation at the shoulder-joint, pleurisy occurs on the same side, or when suppuration occurs in the knee-joint on the same side after fracture of the leg in its lower third. In other cases we may be justified in assuming that a part already diseased, or previously disposed to inflammation in consequence of the general febrile condition, becomes acutely diseased. It sometimes happens, for instance, that an already tolerably firm, quite normally formed, subcutaneous fracture-callus, say of the radius, begins to suppurate in the third or fourth week, if the patient be attacked by pyæmia from a compound fracture of the leg, or from a bedsore. But there still remain a great many cases in which such explanations as those just mentioned do not apply. We then try to content ourselves with the assumption that a disposition to inflammation, especially to the formation of pus in certain organs, is necessarily connected with the suppurative poisoning, and that the purulent poison in the blood acts phlogogenically upon particular organs. I can give you no further explanation in reference to this question, but I should be glad to render this hypothesis more plausible to you by comparison with analogous observations, I mean, namely, by comparison with the specific phlogogenic action of certain drugs, to which I have already alluded when speaking of the ætiology of inflammation, and especially of toxico-miasmatic causes and their mode of action.

Various attempts have already been made to explain this mysterious action of different substances circulating with the blood. Thus Samuel believes that the secret of the specific action of such sub-

stances consists therein that they are retained mechanically in the capillary vessels of certain organs, and thus develop their phlogogenic action in those organs, but it appears to me that the difficulty is not thereby overcome, for it still remains as obscure as ever why cantharidine should be retained in the capillaries of the kidney, mercury in those of the parotid, septic poison in the spleen, croton oil in the capillaries of the intestines, &c.; moreover, it would be necessary, first of all, to explain the reality of this retention.

On the whole, the occurrence of *diffused* metastatic inflammations in internal organs is amongst the rarer phenomena, unless we reckon as such the diffused enlargement of the spleen, which is, indeed, pretty frequent if not constant in pyæmia.

The diagnosis of metastatic abscesses and inflammations is easy when these abscesses are situated on the surface of the body and on the extremities; metastatic meningitis and choroiditis are also comparatively easy to recognise. The diagnosis of metastases to the lungs may be difficult, since these nests are frequently so small and so scattered that they cannot be detected by percussion. Pleuritic effusion often facilitates the diagnosis of metastatic abscesses in the lungs; if bloody sputa and bronchial catarrh are also present, the diagnosis may be made with certainty. The subjective symptoms are often strikingly unimportant; a considerable degree of dyspnœa only sets in with extensive pleuritic effusion. Icterus frequently becomes developed to a greater or less extent in pyæmia; whether the colouring matter of the bile is formed in the blood from the red colouring matter of the latter, without the aid of the liver, or whether icterus cannot occur without such aid, is not yet certain, although the majority of modern observers are of opinion that icterus is always of hepatogenic origin. In any case, icterus occurring in pyæmia does not furnish a diagnosis of abscesses in the liver; the existence of them may be assumed with probability when there is much pain in the neighbourhood of that organ, but it has already happened to me to see in such cases that instead of the abscesses expected, I found acute diffused softening of the liver, combined with almost bronze-coloured icterus. Enlargement of the spleen may be detected by percussion. A considerable amount of albumen in the urine, with epithelial and gelatinous casts and admixture of blood, especially when there is a great diminution in the quantity of the urine, justifies the assumption of an acute metastatic nephritis; whether the kidney is then studded with numerous metastatic

abscesses or is in a state of diffused inflammation, which also sometimes occurs metastatically, cannot be ascertained with certainty during life. Most frequent are abscesses in the lungs and spleen and metastatic inflammations of joints, much more rare are abscesses in the liver and kidneys and metastases to all the parts mentioned formerly.

There is one symptom of pyæmia which we must consider more minutely, namely, *rigors*. They occur irregularly, seldom in the night, but at any period of the day, and their duration and intensity vary immensely; the patient complains at one time only of a slight feeling of cold and transient shivering, at another he trembles violently and his teeth chatter as if he had the ague. At first the rigors occur less frequently, then more frequently, twice or three times a day. Towards the end they decrease. The attacks themselves resemble those in ague as regards cold, dry heat, and sweating; after the attack, however, no complete cessation of the fever occurs, but some fever almost always remains. Now, what actually takes place with the rigors? When we have an opportuity of making observations on ourselves under such circumstances, we feel a peculiar convulsive dragging in the skin; we are compelled, against our will, to strike our teeth spasmodically against each other; if this ceases for a moment we do not feel cold, but rather hot, and the feeling of cold lies more in the imagination, because we otherwise experience such sensations and such convulsive trembling only from the action of a considerable degree of cold. On feeling the extremities and surface of the skin during the rigors we find, indeed, some diminution of the temperature, because the contraction of the muscles of the skin drives the blood out of the capillaries. But if you take the temperature of the body with the thermometer from the commencement of the rigors, you find that the temperature rises constantly and very rapidly, sometimes 3·6°—5·4° Fah. in ¼—½ hour. After the rigors and during the period of dry heat the temperature of the body usually attains its highest point; it may reach 107·6°, but seldom rises much above 105·9°; from that point it gradually falls again. The rapid rise in the temperature of the body no doubt stands in relation to the rigors; moreover, a certain irritability of the nervous system also appears necessary for its production, since in torpid or narcotised individuals rigors occur much more rarely than in very excitable subjects.

Acute diseases of the most various kinds commence with rigors,

especially the acute exanthemata, pneumonia, lymphangoitis, &c., more rarely the infectious, miasmatic diseases, such as typhus, the plague, cholera. These rigors are not generally repeated, however, but only the first outbreak of the disease is accompanied by this symptom. It appears as if the first introduction of certain phlogogenic matters into the blood in otherwise healthy individuals especially predisposes to rigors, or as if certain infectious matters, when they enter the blood, excite intense fever with rigors. If, therefore, we cannot regard rigors in themselves as characteristic of pyæmia, their frequent recurrence and the *intermitting* fever type are still peculiar to this disease. We see something similar in ague only: we there have intermitting accessions of fever, with regular intervals; upon what these intervals depend we do not know, but I am inclined to regard the propulsion by fits and starts of products of disease from the spleen as the immediate cause of the accessions of fever. We have anatomical proofs in melanæmia and metastases of pigment that certain matters pass from the spleen into the blood. We know that accumulations (charges, Schiff) of normal secretions occur in the pancreas and spleen, and are poured out by fits and starts during digestion, and it therefore does not appear to me too bold to assume that with these physiological discharges of certain matters from the spleen pathological products also pass into the blood. Thus, I believe, septic matters, formed, perhaps, from decomposing pus, are poured from time to time into the blood, and, under favorable circumstances, accessions of fever, with rigors, occasioned thereby. An extensive, advancing inflammation around the wound must be regarded as the chief source of such repeated purulent infection. Destruction of the granulating surfaces from repeated injuries to the wound, rapid decay of the granulations from chemical influences, any progressive inflammations set up anew in the wound, may admit the pus into the previously closed lymphatic vessels and veins; then, with fresh inflammation, suppurative decay of the coagula in the lymphatic vessels and the passage of this pus thence into the blood may occur. It sometimes happens, also, as mentioned formerly, that in cases of thrombosis of the veins the central portion of coagulum which confined the pus in them is torn away, and this pus carried by a collateral venous branch into the blood. Lastly, the metastatic inflammations, whether resulting from embolism or without it, may also occasion fresh accessions of fever. That this is not the only source of them is shown by the

fact that we examine, after death, rare cases of intermitting purulent fever, in which rigors were observed ten or twelve times, but no evidence of metastatic inflammations was found; the cause of the repeated rigors may then have lain in the mode of extension of the local process, or have been concealed in the bone or elsewhere. Statistics speak strongly in favour of the assumption that the rigors depend upon constantly renewed processes of inflammation, for it can be shown that the rigors (or at least the intermitting accessions of fever, which may also run their course without rigors) occur much more frequently in persons in whose bodies inflammatory processes of internal organs are recognisable after death than in those in whom this is not the case. It should be distinctly borne in mind as an observed fact that rigors occur almost exclusively at the commencement of acute inflammations, and intermittingly only in ague and *purulent absorption*, while they are wanting in acute septicæmia. The *chemical qualities* of the infectious agent also probably play an important, hitherto unknown part therein. We cannot, unfortunately, learn anything from experiments here. I have never succeeded with rabbits, dogs, or horses, in causing rigors or intermitting accessions of fever by the injection of putrid matter or pus. Pus and ichor act upon animals in the same way as regards fever: only by repeating the injection can we produce in them the intermitting form of fever.

You will understand from what you have just heard that the usual method of taking the temperature morning and evening can give no distinct idea of the course of the fever in pyæmia. Since the observation must thus be made sometimes at the acme, sometimes at the subsidence of an accession of fever, sometimes at the period of the remission (a complete intermission seldom occurs in pyæmia), we thus naturally get extremely irregular fever curves. If we would obtain a perfectly correct outline of pyæmic fever, it would be necessary to leave the thermometer always *in sitû*, and to take the temperature every fifteen minutes; but since this would disturb the patient very much, and other indications enough exist for prognosis and treatment, I have never yet been able to make up my mind to do so. The inquiry whether special matters exist in the pus of pyæmic subjects, or whether the qualitative composition of their pus differs from that of others who recover without any intercurrent attacks, has not as yet led to any results. Neither has the pus of pyæmic subjects always a bad smell, nor do we find in all cases

cocci in the pus of these patients, but the cases in which decomposed pus containing cocci enters into the circulation at the wounds are by far the most frequent. Whether the pus-coccus then grows further in the circulating blood is not known. I have not found cocci and bacteria in the blood of pyæmic any more than in that of septicæmic subjects, and must refer you in regard to this point to what has been said already.

The mode in which pyæmia makes its appearance varies in many respects. This disease, which we look upon as a peculiar malignant form of purulent fever, begins at the time when suppuration commences, or later if fresh inflammations supervene in the wound, either in immediate continuation of the traumatic inflammations, or incidentally later on after the traumatic inflammation-nest has already become circumscribed. The pyæmic fever then becomes developed from the wound-fever or from the secondary fever, and these are regarded by many observers in such cases as prodromal stages of the pyæmia. *The moment at which the patient becomes pyæmic can no more be fixed exactly than that of the transition of primary wound-fever to septicæmia.* I adhere for the present to the term "pyæmia" for the disease just described, and have pointed out to you the absorption of pus as cause, the intermitting course of the fever with rapidly increasing marasmus as chief symptom, and the metastatic inflammations as very essential anatomical phenomena; but it is often very difficult to decide whether we must regard a given case as severe wound-fever or septicæmia, or as severe purulent fever or pyæmia. The rigors may be wanting, and the intermitting course of the fever is then difficult to ascertain, or it may be impossible to diagnose the metastases during life. You have a case of osteomyelitis with very frequent rigors; the patient dies, and you find no metastasis: was it pyæmia? For the beginner, who wishes to have everything beautifully systematised, these questions with their uncertain answers are always discouraging; you will meet with some surgeons who call such cases pyæmia, others who speak of them simply as intense purulent fever, or as febrile marasmus. If you adhere to the description given formerly, and have rightly understood the infection in its relation to venous thrombosis and embolism, it may be hoped that you will not have any difficulty about the name. It is, in fact, scarcely possible to find a name for every combination which occurs between sepsis, purulent infection, diffused metastatic processes, thrombosis, embolism, &c. We see, for instance, sepsis without any trace of

metastases, sepsis with diffused metastases, sepsis with thrombosis and embolism. We see purulent infection without any trace of metastases, purulent infection with diffused metastases, purulent infection with diffused metastases and thrombosis, with thrombosis alone, with thrombosis and embolisms. We have thrombosis with consequent local phenomena without embolisms, with embolisms, with hæmorrhagic effusions, with apoplexies, &c. In addition to the terms already in use, some others have been invented to designate combinations of the various processes already mentioned; for simple purulent infection—infection with thin unhealthy pus, ichor—Virchow wishes, as I told you before, to introduce the name *ichoræmia*. O. Weber employs the expression *embolæmia* for the cases in which emboli are found in the blood. Very practical appears to me the classification which Hueter has adopted in his excellent work on this subject. He calls the disease in cases of simple purulent infection without metastases *pyæmia simplex*, and with metastases *pyæmia multiplex*.

It is only within the last ten years that the differentiation between "septicæmia" and "pyæmia" has come much into use; it is based upon ætiological, clinical, and anatomical phenomena, as I have already explained to you. Voices are already raised against this differentiation; it is asserted that the poisoning agent is always the same in wound-fever, septicæmia, and pyæmia, and is always the product of the proliferation of cocci. I can assure you that we know nothing on that point; perhaps it is correct, perhaps not. The clinical features of the above-named conditions are, however, sufficiently varied in most cases to keep them separate for the present; if it should once become evident that the difference depends merely upon the more or less intense action of one and the same chemical process, this will be a great scientific gain, but it will not diminish the clinical, and especially the prognostic, value of the forms of disease described above. That there are cases to which the term "septo-pyæmia" proposed by Hueter is very applicable, *i.e.* in which the clinical phenomena of septicæmia and pyæmia merge into each other, is confirmed by my experience. The term "peracute pyæmia," employed by earlier surgeons—*e.g.* Stromeyer—corresponds to the modern expression "septicæmia." What the French call *gangrène traumatique fondroyante* is a rapid, progressive sloughing of limbs in living subjects, with great development of gases even deep amongst the muscles, and green discolora-

tion. It is very rare, and I have seen as yet only two such cases after amputation of the thigh for severe injury.

As regards the *course* of purulent infection, it is for the most part an acute one (8—10 days), frequently subacute (2—4 weeks), seldom chronic (2—3—5 months). The acute cases run their course so rapidly, partly from the intensity and frequent repetition of the infection, partly from the extensive metastases. In the chronic cases it is usually only a question of a moderately intense infection in strong or very tough individuals, which is not renewed often, and of metastases to external parts, abscesses in the cellular tissue, and suppuration of joints, by which the patients are kept in a state of bad health after the other consequences of the purulent infection have disappeared. The *prognosis* depends essentially upon the course which the disease runs. The more frequently the rigors recur, the more rapid the loss of strength is, and the earlier symptoms of internal metastases present themselves, so much the sooner will the patient die. The longer the intervals between the accession of fever are, the better the strength keeps up, and the longer the tongue continues moist, so much the more hope is there that the patient may recover; he is not out of considerable danger until the wound looks quite healthy again, and he has been entirely free from fever for several days, and otherwise presents the appearance of convalescence. It very rarely happens, unfortunately, that a patient who presents *all* the symptoms of distinct pyæmia above mentioned recovers.

We must now return once more to the *ætiology* of traumatic infectious fevers. That they result for the most part from the absorption of products of inflammation, from ichor and pus from the wound or from the inflammation-nest, now scarcely admits of any doubt; that they *always* so arise is, indeed, disputed by many. There are surgeons who assert that pyæmia also arises from a *miasma*, and, in fact, from a *miasma* which becomes developed in sick-rooms from the wounds of many patients lying together in them. This view is based chiefly upon the fact that, when several severe surgical cases (in large hospitals, and especially in military hospitals) are lying together, many of these cases prove fatal from pyæmia, and that slighter cases also, *e.g.* patients with cicatrising granulating wounds, become pyæmic under such circumstances. This is not the place for polemics, and I must therefore content myself with explaining to you the position I take in reference to

this view. I can by no means admit the miasmatic origin of pyæmia, if by miasma is understood what I understand by it, for the cases here in question and for many others, namely, dried component parts of pus and ichor in the form of dust, perhaps also in the form of very minute organisms combined therewith, which are suspended in the air of badly ventilated sick-rooms, or adhere to the walls, the bed furniture, the dressings, or badly cleaned instruments. These in many respects differently constituted bodies, most of which possess phlogogenic properties, will naturally accumulate most where there is most opportunity for their formation and adhesion, therefore in ill-ventilated wards, with hurried attention to the sick, with want of proper cleanliness, and when patients remain constantly in the same rooms. Whether every kind of pus, moist or dry, is equally deleterious it is impossible to say; experiments on animals have given no information on that point. I regard the notion of living and dust-like miasms as a very fruitful one, and if new ideas are thereby called forth in one of you which lead to further study, one of the chief objects of my efforts as a teacher will have been attained. The old theory of miasms in a gaseous form has always led us into fresh difficulties; many intelligent people have puzzled their brains with it, but without much good result.

Another much ventilated question is the following: *Is pyæmia contagious?* The answer to this question comes, in a certain sense, of itself affirmatively and negatively from the notion I have just given you of the pyæmic miasma. A fixed miasma in the form of dust which comes from a suppurating, pyæmic patient must, at the same time, be spoken of as a fixed contagium; but this miasma may, according to my view, come equally well from a patient not pyæmic. This cannot, indeed, be called a contagium in the meaning of the specificists, for whom a contagium causes always the same disease only. You see that the dispute concerning the contagiosity or non-contagiosity of pyæmia must always reduce itself to the fundamental assumptions as to the nature of the disease; it is of importance for those surgeons only who regard pyæmia as a specific disease of an entirely peculiar kind, not connected with the purulent fever, an assumption I consider to be unfounded and practically useless, and against which I have fought for a considerable time, not, I hope, without success. With all this is always connected the question *whether the pyæmic infection enters the body through*

the wound only, or through the skin and mucous membranes. Although the latter is not impossible, I have not yet made any observation by which such an assumption would be proved or even rendered probable, but must rather conclude from my experiences that the infection of the whole body takes place *from the wound only*, whether the respective poison finds in the wound and its neighbourhood the conditions required for its production, or whether it is imported ready formed into the wound from without. In this view I am not rendered dubious by those rare cases even in which no changes or only very slight ones are visible in the wound at the commencement of pyæmia, since the infecting substance may perhaps possess very slight phlogogenic properties, and may, therefore, enter through the wound into the blood and there excite violent phlogogenic action, although the wound was not affected by its entrance. *Sex* appears to have no particular influence upon the frequency of the infectious diseases here in question; rather perhaps has *temperament*, the energy and frequency of the contractions of the heart and arteries, an influence upon the absorption of the deleterious matters. Judging from general impressions, children appear to be less disposed to pyæmia than adults. To ascertain this from statistics is extremely difficult, because so few severe injuries occur in women and children as compared with men; that, consequently, the number of men who die of traumatic infectious fevers is much larger than that of women and children proves nothing for the predisposition of one or another class of individuals to these diseases. *Open* wounds of bone predispose especially to pyæmia; according to calculations from my experience, injuries to the lower extremities are followed most frequently by pyæmia, injuries to the trunk most rarely. The *time of year* and the *crowding of severely injured persons in hospitals* has, according to my experience, only an indirect effect, if any, upon the production of pyæmia, since the infectious matters in the dressing materials, &c., thereby become accumulated in greater quantity, and the opportunities of infection rendered more frequent.

Lastly, I must speak of the so-called *spontaneous pyæmia*. We meet with cases in which multiple abscesses, of the subcutaneous cellular tissue, for instance, or thromboses in the veins with embolic metastatic abscesses occur, although we cannot point out clearly the existence of any primary suppuration-nest. These cases, especially if they run an acute course, are then called

spontaneous pyæmia. There appears to be no good reason for propounding a new theory for these rare cases, in which only the demonstration of the existence of the primary inflammation-nest is wanting; I have no doubt that we shall hear constantly less of these illnesses which, according to the earlier theories, had something very mysterious about them, because we are always learning how to observe more exactly and shall generally discover the connection between the phenomena by diligent research.

With the close connection in which, according to the above view, wound-fever, septicæmia, and pyæmia stand to each other, we are justified in speaking of their *treatment* also conjointly. This is divisible into the prophylaxis and the treatment of the developed diseased conditions; the former is by far the most important part; the object there is to prevent everything which may facilitate the development of those diseases. During the operations themselves, already, there is much to be attended to; all the instruments used, the hands of the operator and assistants, the sponges (which should either be dispensed with altogether and replaced by moistened compresses, or should be quite new) must be thoroughly clean and well disinfected with carbolic acid before they are used. The hæmorrhages must be stopped very completely, especially if sutures are to be used in deep wounds. For the escape of the first, most deleterious secretions very careful provision must be made by the form given to the wound after operation, by special escape-openings arranged immediately, and by the introduction of drainage tubes. If we wish the wound to heal by suppuration, the compresses to be laid on should be steeped in a weak solution of chlorine. As regards injuries from accidents, all deeper wounds, especially all contused wounds, must be kept at rest by means of bandages; what is necessary in the case of compound fractures with wounds has been stated formerly; everything which might excite subsequent secondary inflammations must be most carefully avoided. The patient must lie quiet and as comfortable as possible; I remind you here of the treatment of contused wounds described formerly. That both wounds and patients must be treated with the greatest care and delicacy in applying the dressings is a matter of course, and the greatest pedantry may here prove highly beneficial; daily dressing of the wounds under spray containing 1—2 per cent. of carbolic acid is also very desirable in hospitals.

Of especial interest are the *hospital-conditions*, to which I can allude only briefly here. If but few amongst you may have the good fortune to be actively employed in civil hospitals, it may happen to each of you to wish to know something about these matters in time of war. Hospitals are naturally established only where no miasmata from the ground already exist. The attention of those who erect one must be especially directed to the situation, to an open space planted with trees surrounding the hospital, and to the placing of inodorous privies in appropriate positions. Of all the systems of ventilation yet introduced, that of Van Heke appears to be the only one which has maintained its reputation; the walls of the whole building are traversed by pipes, each of which opens into a sick room. All these pipes start from passages running crosswise beneath the building, at whose points of intersection a kind of windmill is placed which is driven by a steam engine, so that fresh air is thus forced continuously into the sick rooms (pulsion system). You may ascertain the extraordinary efficiency of this system of ventilation at the different seasons of the year in the opera-house here in Vienna. If we have no artificial arrangement for ventilation we must do the best we can with the so-called natural ventilation, *i.e.* we cause to be made above and below in the doors and windows of the wards corresponding ventilation-openings, so that the patients in their beds may be affected as little as possible by the draught. A distinguished English surgeon, Spencer Wells, has said, "there is only one efficient arrangement for ventilation—the impossibility of shutting doors and windows." Equally important with the arrangements for ventilation appears to me to be the judicious use of the wards. No ward should be occupied for more than four weeks continuously. It should then be cleared for a few days and cleaned most carefully; the walls should be painted with oil-colour, to admit of their being washed easily, or should be whitewashed twice or three times a year, or oftener if necessary; the beds should be aired, beaten, and put into the sun and the straw in the bags renewed frequently, but it is best not to use straw- bags at all. Every surgical division should have one or, still better, two supernumerary wards, to admit of a regular change. For this purpose, there should not be more than six or eight beds in a ward, so that a sufficient number of patients can be sent out each week to empty a ward; the new patients should then be put into the ward last cleaned. To obtain

the most favorable results possible in a hospital there must be plenty of room and adequate funds for attendants, washing, &c. In this way even badly arranged hospitals may be rendered serviceable. Large wards with twenty or thirty beds, which cannot be emptied when desirable, on account of a press of patients and for other reasons, are in the highest degree unpractical. The director of a surgical division ought, above all things, to have at his disposal a large number of well-ventilated rooms of moderate size, the emptying and cleaning of which is undertaken on fixed principles. We may now, I think, take it for granted that the worst infectious matters are inodorous, but it would be a great misfortune if the conclusion were drawn therefrom that stinks in wards are innocuous. Bad air will always be dangerous for the healthy as well as for the sick. Bad hospitals, especially badly cleaned wards for surgical patients, are worse than the most miserable dwellings of the poor, and may become murder-dens for the wounded, through a combination of vitiated air with an accumulation of infectious matters. Let surgeons never lose sight of the fact that they are themselves, in many cases, more or less in fault if their patients are attacked by erysipelas, hospital gangrene, pyæmia, &c.; for if everything were still to be put to the account of ever present, invisible and impalpable miasms, epidemic influences, and the constitutional condition of the patients, it would be the death of all progress in our art!

We now come to the treatment of wound-fever, septicæmia, and pyæmia, concerning which it is to be remarked that it is not usual to employ anything against simple wound and suppurative fever of ordinary severity except cooling drinks, fever diet, and a little morphia in the evening, to secure rest during the night. If the fever continues longer than usual, or assumes a peculiar character, febrifuge remedies may be employed. Digitalis is of little use here, on account of its tardy and uncertain effect. Veratria lowers the temperature, it is true, but appears to do little good in the toxico-traumatic fevers; but further observations on that point are desirable, especially as regards pyæmia. According to the minute study of this remedy by Biermer, very especial care appears necessary in using it. Aconite was formerly very strongly recommended by Textor in pyæmia. I have not seen any good effects from that drug. Quinine is the most efficient remedy against the intermitting suppurative fevers, especially in combinatiom with opium: gr. viiss—

xv of quinine in the course of the afternoon, with 1·2 grains of opium in the evening, very frequently stop the rigors. I have employed these remedies with success in severe suppurative fevers, but have found them of less use in well-marked pyæmia. Liebermeister came to the conclusion, after careful study, that quinine only exercises its anti-febrile influence in typhus and other infectious diseases when given in doses of fifteen grains. Observations are also not wanting concerning remedies intended to act directly against the blood-poisoning. The antiseptic internal remedies, acids, solution of chlorine, the sulphates of the alkalies (much praised by Polli), have appeared to me utterly inefficient. We may also employ still other remedies for the purpose of eliminating the organic poison in the blood by means of an increased conversion of tissue. When we observe the profuse diarrhœas which occur in dogs whom we have artificially rendered septicæmic, and who not unfrequently recover after these diarrhœas, we might conclude that the poison is most easily eliminated by the alimentary canal. Breslau has, in fact, seen good results from the repeated administration of laxatives in puerperal fever. Unfortunately, I cannot say the same in reference to pyæmia: profuse diarrhœa in pyæmic patients is generally a serious complication, which leads quickly to collapse. We might also think of promoting the activity of all the secretions by repeated emetics; but this is followed by such great collapse that we must use these remedies very cautiously. I have repeatedly attempted to produce free perspiration in septicæmia, if the skin was dry. This may sometimes be effected by means of a warm bath of an hour's duration, and by wrapping the patient afterwards in warm blankets. This sometimes brings improvement and I even believe that I have saved the lives of certain patients thereby whose cases appeared hopeless according to my previous experience. This treatment deserves further investigation. Free diuresis may be effected by giving a large quantity of fluid, but this appears to have but little effect upon the general condition of the patient.

Lastly, we might hope to cut off the further supply of deleterious matters from the injured or inflamed part by means of amputation, when possible to perform it in sound parts, even if symptoms of severe constitutional affection have already shown themselves. In acute cases of septicæmia and pyæmia this is very rarely followed by permanent good effects, although temporary improvement almost always takes place. In subacute and chronic pyæmia, amputation

may really save the patient's life, but these cases are, unfortunately, rather rare.

We now come back to the idea propounded at first, that very much may be done to prevent the occurrence of severe wound and suppurative fevers, but that, on the other hand, the treatment of these diseases, when they are fully developed, affords but little prospect of success. The reason of this appears to lie chiefly in the circumstance that the septic matter, when once taken up into the blood, acts as a ferment upon it, and thus a small quantity suffices to throw the whole of the blood and all the fluids of the body into a state of putrid fermentation. As I have already stated, I do not regard this hæmato-zymotic action of the septic poison as having been proved. I am rather of opinion that the septic poison, like the diphtheritic poison, the poison of gangrene of the spleen, and similar matters, acts so long and so variously in the body, even when taken up by it in small quantity only, *precisely because the human organism (as well as that of many animals) throws off this poison with great difficulty*, and because it often produces, at the points at which it is retained in the organism, fresh nests of disease in which the poison is reproduced, although perhaps only in decreased intensity. Dogs, for instance, can, in my opinion, bear so much septic poison because they eliminate it with such extraordinary rapidity through the alimentary canal; they overcome in this manner even very severe putrid infections. The capability of eliminating more or less quickly the infectious poisons taken up may, within certain limits, vary greatly in human individuals. I consider this view as a very fruitful one in reference also to typhus, cholera, and the acute exanthematous diseases.

LECTURE XXVII.

4. *Tetanus;* 5. *Delirium potatorum traumaticum;* 6. *Delirium nervosum and Mania.*

APPENDIX TO CHAPTER XIII.—*Of poisoned wounds: stings of insects, bites of snakes; infection with the cadaveric poison (Leichengift).—Glanders. Gangrene of the spleen (Milzbrand).—Foot-and-mouth disease. Hydrophobia.*

THE group of diseases which belong to the traumatic and phlogistic states of infection and still remain to be mentioned includes *tetanus, delirium tremens,* and the very rare *psychical derangements* after injuries and operations. The most varied views are held concerning their origin. Since it is a question of processes which, from their symptoms, must be referred to irritation of the brain or spinal cord, the cause of them has generally been looked for in the nervous system itself. But it is known that by blood-poisoning, with strychnia for instance, convulsions, with alcohol, psychical derangements (intoxication) may be produced, and it thus becomes possible to imagine that there arise the forms of disease from poisoning with peculiar substances which I am now about to describe, which substances *perhaps very rarely and under entirely peculiar circumstances* become formed in the wounds, and absorbed from thence, while in delirium tremens a series of the usual pyrogenic elements suffices to occasion peculiar derangements in the organism already poisoned by alcohol, namely, a fever with predominant psychical derangements of a peculiar kind. The symptoms with which we shall become acquainted in these diseases are also all present in ordinary fever, although in a much slighter and less prominent degree; rigors have an undoubted resemblance to trismus and tetanus, and well-marked psychical derangements, amounting to accessions of mania, are observed partly as so-called fever delirium in many cases of septicæmia, but especially in typhus. In the

description of the individual diseases we shall return occasionally to these considerations, for which we have, unfortunately, no experimental basis.

4. *Trismus and Tetanus.*

This disease, which consists partly in contractions of the muscles of the jaws only (*trismus*), partly of all the muscles of the body (*tetanus*), and in which at one time the extremities, at another the muscles of the anterior or posterior surface of the trunk are more affected, sometimes occurs after wounds, although rarely in comparison to the incidental wound-diseases already spoken of, and still more rarely in persons who have not been wounded. Years may pass in a large hospital without the occurrence of a single case, while at certain times several cases occur in quick succession, so that we are inclined to suspect the existence of some epidemic cause. The disease is by no means confined to hospitals, but before we enter upon these ætiological conditions, I will attempt to give you a short sketch of an acute case.

On the third or fourth day after an injury, seldom earlier, often later, you find that the patient does not open his mouth properly when speaking, and complains of tearing, dragging pain and stiffness in the muscles of mastication. In very acute cases, this first symptom is already accompanied by high fever; in other cases, you find the patient free from fever during this stage. The patient's features gradually assume a peculiar fixed expression, since the muscles of the face are partly in a state of convulsive contraction. Tetanic convulsions then soon supervene, sometimes more in the trunk of the body, sometimes more in the extremities, which occur in accessions of several seconds or minutes duration, and are occasioned by any external irritation, just as in hydrophobia. These convulsions are accompanied by violent pain. Some groups of muscles occasionally remain from first to last uniformly contracted, but without pain, and in many patients the twitchings are entirely wanting, and only a continuous contraction of more or less extensive groups of muscles occurs. The body is not unfrequently bathed in perspiration, the patient's mind quite clear; the urine sometimes contains albumen; the fever often attains a height seldom reached—more than 107·6°. I have seen cases of trismus, however, which rapidly proved fatal, but ran their course without any rise of temperature; and similar

cases have also been observed by Rose. Death may occur within the first twenty-four hours from the commencement of the disease but the patient's condition may go on with considerable severity for three or four days and such cases still be reckoned amongst the acute ones. There is, further, a more subacute or chronic form of trismus alone, or of trismus and tetanus, in which only a moderate degree of trismus is developed, and contractions occur which extend to some groups of the muscles of the injured limb, unaccompanied by pain. There is generally no fever present in these chronic cases. It rarely happens, on the whole, that an acute case passes into a chronic course.

All the symptoms which present themselves indicate that we have to deal with an irritation of the spinal cord and of the motor portion of the fifth nerve. The features of the disease present a resemblance, distant, indeed, to those which we can produce artificially by poisoning with strychnine. Unfortunately, the results of *post-mortem* examinations of these patients are, for the most part, very unsatisfactory. In cases running a very acute course, especially, nothing is to be found in the spinal cord; in cases of some days' duration, Rokitansky states that he has found in the spinal cord a development of fresh connective tissue, from which it would appear as if we had to deal with an inflammatory process in this nerve centre. My investigations of the spinal cord and nerves in tetanus have, as yet, given negative results only. In preparations which were made from transverse sections of the spinal cord by distinguished specialists in the investigation of the central nervous system (Dr. Goll, in Zurich, and Professor Meynert, in Vienna, and kindly shown to me), I certainly saw at many points of the cord the portions of connective tissue strikingly developed; but as this was not combined with an accumulation of fresh cells, it always remained doubtful to me whether this increase of connective tissue really resulted from new formation, and not rather from accidental thickening, which might, indeed, have existed during life. The symptoms in the living subject in an actual case of demonstrable inflammation of the spinal cord are so different from those of tetanus that it also thereby becomes improbable that the latter should result from a neuritis ascendens, leading to myelitis spinalis. That we find here and there in the muscles, and also in the sheaths of the nerves, small extravasations of blood after death, is of little significance for the nature of the disease, since these may have resulted from the

laceration of the capillary vessels in consequence of the violence of the muscular contractions.

Concerning the origin of this disease many theories exist, as is usually the case with all such processes as present no tangible, pathologico-anatomical basis. At first it appeared natural to direct investigation to the nerves, and a number of cases presented themselves in which nerve branches were observed to be lacerated, contused, or irritated by foreign bodies in cases of accident. I myself have seen a few such cases, amongst which, some years ago, was a sporadic case in which, with a comminuted fracture at the lower end of the radius, the medium nerve was half torn away, and on the third day trismus and tetánus suddenly set in, which proved fatal within eighteen hours. It is useless to form theories as to the reason why precisely this kind of injury to a nerve is followed by tetanic contractions, while they very seldom occur after simple sections of nerves, because a whole series of cases exists in which tetanus becomes developed, partly after simple wounds of the skin, partly with fully formed granulating surfaces beginning to cicatrise, or even after the application of blisters, or the stings of bees, and the like. It is remarkable, however, that the disease becomes developed with especial frequency after injuries to the extremities, especially to the hands and feet, while it is seldom observed after much more serious injuries to the limbs higher up, or to the trunk of the body. I have observed, further, that those cases in which the tetanus occurred in wounds already granulating run a milder and more chronic course than those in which the disease developed itself soon after the injury. Rose is of opinion that tetanus occurs especially with wounds which are treated badly, or not all. My experience does not tend to confirm this.

After appeals had been made in vain to the nerves, and even to the tendons, recourse was had to the influence of different temperatures, and some observers came to the conclusion that a hot, close temperature especially favoured the development of tetanus. I cannot altogether disagree with this view, since I have hitherto only seen an accumulation of cases of tetanus with a high, close, thunder temperature, although epidemics of tetanus have been observed in winter. Others throw the blame chiefly upon taking cold from draughts, or upon rapid changes in the temperature of the air in general, as Heinecke has done again recently. Others, again, do not believe that the nervous system is affected primarily, but that

the blood first becomes diseased, and acts only secondarily upon it. Roser has brought into light again, not long ago, the old theory that tetanus is to be regarded as a primary blood disease, analogous to hydrophobia. It cannot be denied that there is a great resemblance between these two diseases. A very striking proof thereof would be furnished if we could produce hydrophobia in animals by inoculation with the blood or secretions of persons affected with tetanus. The inoculation of human beings is, of course, out of the question. I incline very much at present to the humoral theory of tetanus as a peculiar poison-disease, without, however, being able to furnish any proofs thereof. In any case the blood of a man affected with tetanus should be injected into a dog, to ascertain whether tetanus is transmissible by means of the blood from men to dogs; and, further, whether such blood acts pyrogenically. Should tetanus occur in the dog operated upon it might be regarded as proved that tetanus is a humoral disease; if the experiment had a negative result nothing is thereby proved, of course, against the humoral origin of tetanus, since the experiment proves nothing more than that the blood of a tetanic *man* does not produce tetanus in a *dog;* it would remain to be investigated whether the blood of a *tetanic dog,* transferred to another *dog,* is equally without effect. The observation that tetanus may be confined to one of the extremities, and even, as I have seen, to the hand alone, speaks, no doubt, for a very local cause confined to the nerves, but we also see strictly localised lymphangoitis, erysipelas, &c. We might also explain the observation that after amputations, for instance, twitchings not unfrequently commence in the stump before the convulsions become general, by assuming that the tetanus-poison is formed in the wound, first produces irritation of the muscles and nerves of the stumps, and then only of the spinal cord. Much, however, remains to be proved in this field.

The high fever in the majority of cases of acute tetanus, and the circumstance that after the death of such patients the temperature still rises, has greatly occupied the attention of pathologists; the interest was still further increased when Leyden, by means of the production of artificial tetanus of the whole body, which he effected by passing strong electric currents through the entire spinal cord of a dog, also obtained very high temperatures of the blood. A. Fick showed that an excess of heat is produced in the muscles under such circumstances, and thence communicated to the blood, so that the rise of temperature observed in the rectum after death is a pheno-

menon of the equalisation of heat between the muscles and the rest of the body. If it can no longer be doubted after these experiments, in which I took part, that the temperature of the body is considerably raised by tetanic contraction of the muscles, it is not proved thereby that in traumatic tetanus in the human subject the high fever temperatures must depend solely or predominantly upon the muscular contractions. The observation that cases of tetanus running a very acute course may, although rarely, terminate almost without fever, speaks against this. In reference to this question, also, there are still many problems to solve.

The *prognosis* is, unfortunately, a bad one in most cases. Of the patients attacked acutely extremely few recover. Of the chronic cases which extend over fourteen days many terminate favorably. The latter cases are, unfortunately, rare.

With our deficient knowledge of the ætiology of this disease we are obliged to regulate our *treatment* of it by the symptoms. A great number of remedies have been recommended at different times. In general, the treatment with narcotics, opium, and chloroform, which I also have adopted, is that most in use. The opium is given in very large doses, up to fifteen grains and more daily; or a corresponding quantity of morphia, which latter is best done by subcutaneous injection. The convulsions sometimes cease after this, but sometimes these remedies have no effect; in any case, the patients experience some relief subjectively therefrom. During the individual accessions, the patients experience great relief from the inhalation of chloroform to complete narcosis. Hydrate of chloral has been given with some success in tetanus, internally up to 60 or 75 drops in a glass half full of water, or per anum once or twice in the twenty-four hours, until a permanent hypnotic effect is produced. Several cases have recovered under this treatment. On the whole, we strive to render the acute course milder, and to convert it into a more chronic one, because there is then more hope of recovery. Of other modes of treatment I will mention the frequent employment of warm baths. Further, the application of strong stimulating remedies along the spine, large blisters, moxas, ferrum candens, remedies from which I cannot promise you any good result. Lastly woorali, which has sometimes been tried of late, but which has not answered the expectations entertained of it.

In the chronic cases you need not adopt any special treatment; the patient must remain in bed, and keep himself perfectly quiet.

We try to defend him from all deleterious influences, and especially from any causes of physical or psychical excitement.

5. *Delirium potatorum Traumaticum. Delirium Tremens.*

We now come to an enemy to the wounded, dangerous, fortunately, to only a few. You have no doubt heard already of delirium tremens, that acute outbreak of chronic alcoholic poisoning, which may occur quite spontaneously, or also with many acute diseases, especially pneumonia. Injuries are a not unfrequent occasional cause of an outbreak of delirium tremens. You will become more familiar with this disease from the lectures on internal medicine, since the accessions, from whatever cause arising, do not differ essentially from each other. I shall, therefore, be brief in my remarks here.

The outbreak of the disease usually takes place within the first two days after the injury, seldom later. Only such patients are attacked by it as have been accustomed for years to the free use of alcohol, especially of schnaps and rum; but it is a mistake to suppose that wine- aud beer-drinkers are safe. Sleeplessness, great restlessness, trembling hands, an unsteady look, tossing about in bed, and great talkativeness, are the first symptoms; then follows the delirium. The patients are muttering constantly to themselves, see small animals, gnats, flies, in swarms before them; from beneath their beds creep mice, rats, foxes, &c.; they fancy themselves to be in a smoky atmosphere, or have the feeling of being tossed up and down. The delirium frequently assumes the most comic forms: a soldier whom I treated for delirium tremens in Zurich saw a great number of other soldiers in his water-glass; when I went into his room he spoke low to my assistants, because he took me for his major, and so on. In general the hallucinations are of a cheerful kind, but the patients are nevertheless tormented by an indescribable restlessness, and throw themselves about constantly in bed and want to run away. Unless we have two powerful attendants at our disposal to control these patients, we sometimes have, unfortunately, no other alternative but to put them on a strait waistcoat and fasten them in bed. At the same time these patients are, for the most part, very good-tempered in their delirium, and if we speak to them very earnestly they give perfectly sensible answers, but fall back immediately into their hallucinations. Of all the kinds of injuries, fractures, and especially compound fractures, most

frequently occasion the outbreak of this disease and until we possessed compact dressings for such injuries, it was a difficult task to fix the broken extremities, since the patients, in spite of the pain caused thereby, moved the broken ends of bone so violently that every dressing with splints became loosened in a few hours.

The *prognosis*, even after delirium has set in, is, in the opinion of most surgeons, not an unfavorable one. The small number of observations I have had the opportunity of making do not lead me to agree with this view. Of the few patients whom I have treated for acute delirium tremens—which disease is rare here in Vienna—at least one half have died; they often collapsed quite suddenly, became unconscious, and died soon afterwards. Others recovered, especially if I succeeded in keeping them asleep for some time, and this is the point to which the treatment must be directed. Opium in large doses is the remedy employed almost universally, to which may be added small doses of tartar emetic. Under this treatment the patients fall eventually in a comatose state, out of which, in favorable cases, they awake cured, but sometimes pass away without waking. I cannot recommend to you any better remedy than opium in delirium tremens, although I must confess that I cannot regard the administration of it in large doses (1½—6 grains every two hours until sleep follows) as free from danger. There are also surgeons, especially in England, who would do away altogether with the treatment with opium and tartar emetic, and recommend a more expectant method. Others have attained good results with digitalis. Most surgeons are very well satisfied with the opium treatment, and the simultaneous administration of strong wine and brandy is recommended. Hydrate of chloral, also, in large doses, has been much praised in this disease quite recently. Somewhat more favorable appears to me to be the prognosis of the more chronic cases of delirium potatorum without maniacal accessions; strong grog (*sic*) does good service in such cases. I give the following mixture: the yelk of one egg, one ounce of arrack, ten ounces of water, two ounces of sugar—one tablespoonful every two hours; it is not an unpleasant drink, and is also useful as an exciting remedy for elderly people. I must warn you against any abstractions of blood, which are dangerous in such patients, and not unfrequently have caused a rapid collapse ending in death.

The results of the *post-mortem* examinations of persons who have died of delirium tremens furnish no particular explanation as to the

immediate cause of death. We find the changes usually observed in habitual drunkards: chronic catarrh of the stomach, a fatty liver, Bright's kidney, thickened membranes of the brain, but nothing constant in the substance of the brain itself.

6. *Delirium Nervosum and Psychical Disturbances after Injuries.*

By *delirium nervosum traumaticum* is understood a state of the highest nervous exaltation without fever after injuries, as it is said to occur especially in hysterical subjects. I have as yet seen only one case to which I should be inclined to apply this name. A man about twenty-four years old, who had never drank much, had delirium without fever after a compound fracture of the leg with a slight wound, like an habitual drunkard. The hallucinations resembled those of delirium potatorum and ran their course under soothing treatment and the use of opium, without maniacal accessions. After four days the delirium ceased, and the patient remained perfectly sensible.

Lastly, I must mention to you those interesting and rare cases in which, after operations upon otherwise perfectly healthy individuals, psychical disturbances become developed, cases which frustrate all attempts to explain them, and find their analogy in the fact that after other acute diseases, also, *e. g.* pneumonia, acute rheumatism, typhus, the development of true mania is observed. I have seen two such cases in the Berlin surgical clinique, in both of which, after a total rhinoplastic operation, melancholia with religious impressions set in. Both patients were Roman Catholics; one, a young man, was constantly tormented by the desire of coming to a distinct conclusion concerning the Trinity; the other, a young girl, sought to punish herself by prayer and castigations for having yielded to her vanity in so far as to have had a fresh nose formed after the entire destruction of the natural one by lupus. The young man had repeated accessions of violent mania. Both patients recovered completely in a few weeks. I have also heard that v. Langenbeck observed accessions of mania after a plastic operation in Berlin, and v. Gräfe and Esmarch after an operation on the eye. On the whole, however, such cases are extremely rare.

Appendix to Chapter XIII.

On Poisoned Wounds.

We have now still to occupy ourselves with certain forms of injury in which, simultaneously with the injury itself, poisons are introduced which cause partly very violent local inflammations, partly dangerous general diseases. Such poisons are well known to be peculiar to many animals; in others they become developed in consequence of certain diseases, and then communicated from these diseased animals to man.

The *stings* of a great number of small *insects* are out of proportion as regards their consequences to the slight mechanical irritation produced. Sometimes, indeed, it may depend upon a special irritability of the skin that some people undergo extensive although transient inflammation of the skin after being stung by bugs, gnats, fleas, &c., while others do not suffer at all therefrom. But a prick with a pin is a much greater injury than the sting of a flea, and yet the latter causes itching and burning of the skin, and wheals, while the former has no effect whatever. It is, therefore, not improbable that, in the case of the stings just mentioned, some irritating substance penetrates into the skin at the same time. The stings of bees and wasps cause, as is well known, much more violent symptoms; an extensive, very painful inflammation of the skin sometimes follows, with much redness and swelling, which usually becomes dispersed, and is not dangerous to the organism, but may be extremely troublesome. A great number of such stings are, however, not altogether without importance; if they affect the tongue, the gums, or the eyelids, a certain amount of danger may be caused by the swelling of those parts. But since these inflammations subside in a comparatively short time, medical aid is seldom sought for. Various cooling remedies are employed to relieve the pain, such as scraped potatoes, cabbage leaves, and the like. For more violent inflammations, fomentations with solutions of lead and other antiphlogistic remedies are used. Still more violent in their effects than the stings of bees and wasps are those of *tarantulas* and *scorpions* in southern lands. There occurs after them a still more extensive inflammation of the skin, with very violent burning pain, and sometimes with the formation of blisters. Fever may supervene, but even this state of things is not generally

dangerous, except from the particular site of the injury. The same treatment holds good as for the stings previously mentioned.

Fortunately we have in our part of the world but few kinds of *poisonous snakes*, and even these are not common. Amongst them may be named the Vipera Berus and Vipera Redii, with two hooked, bent, poison-teeth, in which run the excretory ducts of small glands which pour their fluid into the wound when the bite takes place. The bite of these snakes is not quite so dangerous as is generally believed. From statistical calculations it appears that out of sixty persons bitten about two die. The pain is very severe; violent inflammation, tension, and swelling of the skin set in, with high fever; a feeling of great anxiety, prostration, vomiting, and sometimes slight jaundice. As regards the treatment, it will be best to suck the wound immediately, which can be done without danger, since the poison is not absorbed by the stomach, or by the mucous membrane of the mouth, and is, perhaps, even destroyed in the former. The wound should be washed out immediately, and the advice is often given to tie a cloth tightly round the limb above the wound, to prevent absorption. In most cases the poison has already been absorbed before the patient is seen by the surgeon. Opinions differ as to the advisability of applying a cupping-glass, or of cauterising, burning, or cutting out the wound, but I should consider it best to cauterise, even in the later stages. The local inflammation of the skin is to be treated chiefly in reference to the painful tension; inunction with oil or grey ointment, and prevention of the access of air by various means with which we become familiar in connection with the treatment of superficial burns. Internally an emetic is given, then antiseptic remedies, *e. g.* mineral acids; ammonia is also said to be of use, and is recommended by American surgeons against snake-bites. Putz injected the contents of a Pravaz's syringe of a mixture of equal parts of liq. ammon. caustici and water under the skin in the neighbourhood of the bite of an adder, and gave twelve drops of the same mixture internally several times a day. The patient, a girl eight years old, who already presented very serious symptoms, recovered. It would be better in future to dilute the liq. ammonia with 2—3 parts of water, because an abscess formed at the point of injection. Of all the snake-bites in southern countries that of the *rattlesnake* is the most dangerous; it sometimes proves fatal in a few hours. The local inflammation of the skin, which is very violent and spreads widely, not unfre-

quently passes rapidly into gangrene, and does so, as far as we can judge from the best descriptions, quite directly, without previous formation of thromboses in the arteries or veins. The tissues which come into contact with the poison become so much changed immediately in their chemical character that they no longer run through the normal stages of conversion, but die at once. The patients die with delirium, coma, and rapid collapse. If a large quantity of poison enters the wound and becomes absorbed at once, death occurs very quickly, before any considerable inflammation has set in. The chief symptoms, then, are cyanosis, dyspnœa, and collapse; sometimes also convulsions, as occurs also in poisoning with prussic acid.

A substance which has a very strong phlogogenic action, and probably varies greatly in its chemical composition, is the so-called *cadaveric poison* (Leichengift). Many of you may already have had experience of this in the dissecting-room. This putrid poison, which becomes developed during the decomposition of the bodies of men and animals, may very well be identical with the much-sought-for putrid poison for which we are looking in tissues in a state of ichorous inflammation, in decomposed pus, &c. If, during the examination of dead bodies, some of the fluid of the tissues enters into small, almost imperceptible wounds in the skin, very unpleasant symptoms may result therefrom; in many cases, such contamination of a wound may have no consequences whatever. The great inequality of the effect renders it probable that the poison is not equally active at all stages of decomposition. Cases were formerly observed frequently, in England especially, in which little pain was felt in the wound at first, but great prostration, headache, fever and nausea soon occurred; then follow delirium and sopor, and, in some cases, death within forty hours. It is asserted that precisely these very bad cases of septicæmia occur most frequently after *post-mortem* examinations made very soon after death in bodies not yet cold, in which cases it appears doubtful whether a product of disease already developed in the living body has not been introduced into the wound in the surgeon's skin, since the condition, at least, which is generally designated as decomposition, and which makes itself known by a bad smell, has not become established. In opposition to this malignant acute form stand those cases in which the poison acts locally only. There occur in the injured finger, in the course of twenty-four hours, moderate pain and slight induration; then a dry scab forms upon the wound, beneath which

there is always pus, although in very small quantity. The scab forms again as often as it is removed, and the part remains painful and hard; after a while, the epidermis becomes thickened at that point, and converted into a painful wart-like nodule, with a moist surface (Leichentuberkel).

Any one who has a disposition to these purely local formations is, for the most part, but little disposed to general infection. Between these two just described per-acute and more chronic modes of action of the cadaveric poison stands a third, in which there supervenes upon the local inflammation an inflammation of the lymphatic vessels and axillary glands, which, if treated early, may be dispersed, but frequently leads to the formation of abscesses in the arm. In rare cases, such a lymphangoitis, with formation of abscess, may go on for months.

As regards the first treatment of the portion of skin infected with cadaveric poison, I advise you, first of all, to pour cold water for a considerable time over the wound and not to check any hæmorrhage which may be present. In a great many cases, the deleterious matter will thereby be completely washed out, and no further infection follow. If there is redness about the wound, then cauterise freely with nitrate of silver or fuming nitric acid; this is, no doubt, very painful, but it has an excellent effect; fresh pus frequently forms beneath the eschar, it is true, and you must remove the eschar and cauterise again until pus no longer forms beneath it. After a pretty large experience in my own person and amongst my anatomical pupils, I do not consider cauterisation immediately after contact with cadaveric poison very advisable. Small lacerated wounds which do not bleed and excoriations are always more dangerous as regards infection than deeper incised wounds, the reason of which is that the blood escaping from the latter carries the putrid poison out of the wound. The susceptibility for cadaveric poison varies, moreover, in different individuals; repeated infections appear rather to increase than to diminish it. If lymphangoitis set in, the arm must above all things be kept at rest by means of splints, and the treatment already mentioned for that disease carried out. You may represent to yourselves the course of things during the appearance of the symptoms just described as follows: a small quantity of fluid from the body (or from decomposed pus of living subjects) enters the wound; the opened

lymphatic capillaries here take up this decomposed matter and carry it into the trunks of the lymphatic vessels; coagulation may now occur in the latter, and the poisonous matter then acts as a specific irritant upon a small district only. In the other case the lymph first coagulates in the nearest lymphatic glands, or the intra-glandular lymphatic ducts become compressed by the great swelling of the glands, and passage through the latter thus prevented In this case also the disease continues local, although extending over a considerable district, and not unfrequently leads to suppuration with fever (as also in other, non-specific inflammations of the lymphatic vessels). Lastly comes the rarest case of all, the poisoned lymph, which now itself acts further as a poison, enters the blood and excites there also to chemical changes, and we have septicæmia from cadaveric poison.

From the cases which recover it may be seen that the deleterious matters formed during the whole process may be eliminated from the body by means of the secretions and excretions; but it is not known exactly in what manner this happens. In many cases some amount of putrid substance becomes completely encased in a lymphatic gland, or in another inflammation-nest, where it may lie harmless and be eliminated gradually later on. By violent movements, however, and the consequent increased pressure of the blood, the poison is again driven into the lymphatic vessels, and excites fresh, acute, local and general infection. If such hard lymphatic glands remain after infection with cadaveric poison, a warm bath daily is the best means of causing rapid elimination of the poisonous matter.

We have still some poisons to speak of which become developed in certain diseases of certain animals, and are communicable from those animals to man. To these belong the *glanders*, *gangrene of the spleen*, *foot-and-mouth disease*, and hydrophobia. Fortunately, these infections are constantly becoming less common in civilised countries in consequence of improvements in the sanitary arrangements, so that you must look upon it as a lucky accident if you have an opportunity of observing one of these diseases in the cliniques during your studies there.

The *glanders* (maliasmus, morve) is an infectious disease which occurs especially in horses and asses, and is communicable to many other animals, but not to horned cattle.

An inflammation of the mucous membrane of the nose, with

formation of nodules of greater or less size is characteristic of this disease. Thick, tough pus is secreted, the nodules become cheesy and break down, and ulcers with a caseous floor are formed; swellings of the lymphatic glands, tuberculoid nodules in the lungs, and general marasmus supervene, and the termination is, in almost all the cases, a fatal one. The more chronic form of glanders, which runs a milder course, is also called "worm." It is more rare, and in it nodules form in the skin which break down gradually and become developed into ulcers. Glanders may run an acute course and prove fatal in ten to twenty days; the chronic form may go on for several months or even a year. The infection passes from one animal to another, partly through the inoculation of excoriated portions of the skin with the secretion of the ulcers, more frequently almost through an evanescent contagium proceeding from the diseased animals, which is taken up by the lungs or intestinal canal. The disease is not always localised first in the nose, but sometimes in the lungs, in which case it is, at first, very difficult to diagnose.

Glanders and worm-disease in animals are communicated to man chiefly by accidental inoculation. If pus from a glandered horse come into contact with a wound or excoriation in a human being, or if very intensely poisonous glander-pus be applied to the sound skin, or to a mucous membrane, very acute inflammations with general septic disease may set in, which generally prove fatal. Cases also occur in which a local infection is not demonstrable and in which infection through the organs of respiration or the intestinal canal is assumed. The chronic form of glanders is rare in the human subject; the chief symptoms are: pustular inflammations of the skin, formation of abscesses and ulcers in various parts of the subcutaneous cellular tissue; the danger here is not so great. In some cases of acute poisoning from glanders, lymphangoitis and suppuration occur which are confined to the injured extremity; in others, a diffused erysipelatous redness, with great swelling of the skin, is developed very rapidly, while at the same time very intense fever supervenes. The local inflammation may pass into gangrene; delirium sets in, soon followed by a state of coma. Diarrhœa and a purulent discharge from the nose generally occur later; pain in the muscles is also sometimes present, and with these symptoms death ensues. The whole disease may run its course in a very short time; thus I remember that, when I was a

student in the Göttingen clinique, I saw a powerful, healthy man who died in a few days from glanders, but it also happens sometimes that patients with acute poisoning of this kind survive ten to fourteen days, and that in them all the symptoms of pyæmia, especially a number of hæmorrhagic *abscesses in the muscles*, are present, *which are so characteristic of glander-pyæmia that we may infer from them the existence of that disease.* The acute, rapidly fatal form of glanders may, in rare cases, develop itself out of the chronic form; on the other hand, the acute form has been observed to pass into the chronic. Men who have much to do with horses are, naturally, especially liable to this disease, which never occurs primarily in human beings. Of the treatment of this form of poisoning there is, unfortunately, but little to be said. We treat the most prominent symptoms as in acute pyæmia. Iodine, arsenic, and creosote are recommended as counter-poisons against glanders.

Gangrene of the spleen (anthrax, pustula maligna) is an infectious disease which becomes developed primarily most frequently in horned cattle.

The disease took its name from the fact that, on examining the bodies of animals dying of it, the spleen was found to be enormously swollen, of a blackish-red colour, and gangrenous as it were; in many cases, moreover, the intestinal mucous membrane is blood-red and swollen; the loose sub-peritoneal cellular tissue, sometimes also the subcutaneous cellular tissue of one or other of the extremities, is œdematous; in the intestinal mucous membrane, and sometimes also in the skin, we occasionally observe infiltrations resembling carbuncle, which rapidly become gangrenous. The disease, like all infectious diseases, runs its course with varying rapidity according to the quantity and intensity of the poison absorbed, and to the power of resistance of the person attacked. The course may be of an apoplectic character, or it may extend over several days. Graminivorous animals are more easily infected thereby than omnivorous or carnivorous animals. The contagium is a fixed one attached to the products of the disease and to the individual affected. Concerning the primary origin of grangrene of the spleen nothing has been clearly ascertained. Since epidemics of this disease occur more frequently in some neighbourhoods than in others, it has been thought to depend partly upon the nature of the soil and of the plants consumed for food, though, perhaps,

secondarily only. The intestinal secretion becomes mixed with the dung of the diseased animals, and has been shown to be an enduring specific poison. If such dung is spread upon fields and eaten fresh, or even dried upon hay, by other animals, the disease may spread in that manner.

The communication of the disease to man occurs most frequently by means of the secretion of the malignant pustules. If that or the dried skin of the dead animal comes into contact with the human skin, the poison may penetrate into a hair-follicle or sweat gland even if the skin be sound. An insignificant looking pustule is first formed, which itches violently and then burns, in the centre of which a vesicle filled with black blood appears; a considerable degree of fever soon sets in. In bad cases, the inflammation of the skin soon assumes the character of a carbuncle and passes rapidly into gangrene; the course of the disease resembles that of malignant carbuncle already described and the disease terminates, if left to itself, for the most part fatally. Internally the well-known antiseptic remedies are given. The anthrax itself should be attacked energetically with incisions, excisions, caustic potash, or fuming nitric acid. If the patient comes early under treatment, and no intense infection of the blood has yet been developed, there is hope of his recovery; if there is full development of the malignant pustule, with symptoms of septicæmia, death is certain. Recent observations have shown that infections with this poison, resulting accidentally in veterinary surgeons from examinations of diseased animals, do not always run such an unfavorable course, but frequently give rise to a phlegmonous inflammation of moderate intensity, which may become dispersed in a few days with scaling off of the skin. Cases have very recently been decribed by Leube and W. Müller, in which, after the eating of the flesh of animals which had died of gangrene of the spleen, violent inflammation of the intestines and death ensued. According to Bollinger, the milk of cows suffering from this disease also infects the human subject. Whether this disease can also become developed primarily in man, whether the malignant carbuncle formerly described always arises from infection, or also spontaneously from similar ætiological conditions as in animals, is still a matter of dispute. Distinguished surgeons and veterinary surgeons have turned their attention to this subject; the attempts to transfer the secretion of malignant carbuncle from men to animals have been very uncertain in their

results; the observations partly contradict each other, and, in short, the relation of these various forms of carbuncle and pustule to each other, in reference to their ætiology, is not yet explained.

The idea that the specific poison of anthrax is connected with certain very minute organisms has quite recently gained ground more and more; Davaine, especially, is of opinion that the bacteria sometimes observed in the blood of living animals affected with gangrene of the spleen, and pretty constantly in that of animals which have died of that disease (first described by Pollender in 1855), are the cause of it. But since it is asserted, on the other hand, that other animals can be infected artificially with blood from animals suffering from gangrene of the spleen, which contains no bacteria, it may fairly be doubted whether the poison cannot exist independently of the latter. In the cases mentioned by Leube, innumerable cocci and bacteria were found in the mucous membrane of the intestines (mykosis intestinalis, Buhl). It is asserted by many that the bacteria found in gangrene of the spleen are of a different kind to those met with in decomposed tissues. Bollinger asserts that small cocci (bacteria-germs) exist in the blood of every animal which dies of gangrene of the spleen, but that they have often escaped observation on account of their minuteness; he regards the vegetation of these fungus-elements as the essential cause of the disease, which is much favoured, no doubt, by the disposition thereto of certain kinds of animals, by their food, and by the nature of the soil and the state of their stalls. My own investigations have confirmed my view that the bacteria found in the blood of animals affected with gangrene of the spleen belong to the meso- and megalo-bacteria, such as are not unfrequently formed in the blood and pericardium of decomposing dead bodies; further, that they rapidly form cocci and frequently resting spores. Frisch, after introducing blood containing bacteria into the cornea of rabbits, observed star-shaped fungi, evidently consisting of bacteria, which developed an enormous power of vegetation and led to suppuration of the eye, but never to general infection nor to the death of the animal. The attempts observed by me to inoculate rabbits, guinea-pigs, sheep, and dogs with the blood of horses and cows which had died a short time before from gangrene of the spleen, showed that the inoculation took effect with the greatest certainty when there was least doubt as to the presence of bacteria in the blood employed for that purpose. Moreover, inconstant results of

the inoculations in many respects presented themselves here as in the case of other observers.

We must also speak of the *foot-and-mouth disease* in horned cattle, since its communicability to man has been established by recent investigations.

In horned cattle the disease consists therein that bladders and pustules form on the mucous membrane of the mouth and at the roots of the hoofs, then on the udders of the cows, which heal spontaneously in the course of five to fourteen days. This has been the usual termination of the disease, which spreads epidemically, partly by means of the secretion from the pustules and of the milk, partly also, as is assumed, by means of an evanescent contagium; although the animals attacked become greatly emaciated, only young calves die of the disease.

The communication of this disease to man takes place by contact of injured portions of the skin with the secretion of the pustules, or by the copious use of *unboiled* milk from the diseased animals. When the disease has arisen in the latter manner, bladders and pustules form in the mouth and on the hands and feet, as in the cattle. Angina and catarrh of the stomach may supervene. The treatment consists in frequent cleansing of the mouth, painting the bladders in the mouth with solutions of borax (five parts to thirty parts of honey), and touching the pustules on the hands and feet with nitrate of silver. The infectious matter in the milk is destroyed by boiling. It is not improbable that many of the aphthous affections in little children arise from infection by the milk of cows suffering from foot-and-mouth disease. The disease runs its course in man with equally little danger as in horned cattle; only very young, weakly children could be in serious danger from it.

Better known and no doubt more frequent than the two diseases just described is *rabies*, or *hydrophobia* (lyssa), which is communicated from animals to man. Bollinger does not admit that the disease is still developed primarily. It is only communicated by the bite of the diseased animals and by the saliva flowing into the wound; the poison affects all warm-blooded animals, and does not lose in virulence by the inoculations, but is always communicable with equal intensity. A mad dog bites a cat, for instance; the disease becomes developed in the latter and it bites a man; the saliva of the man, when he has become affected with the disease, is communicable by inoculation to another animal, and so on. The

disease may also be produced by inoculation with the blood of rabid animals.

The symptoms in dogs are described by veterinary surgeons as follows:—A violent and a quiet form of madness are distinguished. Before the outbreak of both forms, the dog is dull and takes little food for about a week; the madness now begins; the dog runs about without an object and with an unsteady look, urged on, apparently, by some inward anxiety, and bites at everything, when irritated, which comes in its way. Lastly, emaciation sets in, the gait becomes unsteady, paralysis of the posterior extremities supervenes, the bark changes to a kind of howl, convulsions occur, and three or four days after the latter symptoms, death ensues. In the quiet form of the disease, paralysis of the muscles of the lower jaw soon sets in, and therewith the incapability of biting and eating. The other symptoms are the same as above. Some observers do not recognise these as two distinct forms of the disease, but only as different stages lasting for a longer or shorter time. The essential phenomena found after death are, according to Bollinger, a black, thick, pitchy appearance in the blood, œdema of the brain, more or less marked catarrhal changes in all the mucous membranes, especially of the lungs and stomach, often with hyperæmia and ecchymoses, hyperæmia and cyanotic colouring of the parenchymatous organs, deficiency of normal food-materials in the stomach and intestine and presence of indigestible foreign bodies in it, and lastly, great emaciation of the entire animal. I do not know whether any microscopical investigations of the brain and spinal cord have been made in this disease, but it is highly probable that in the cases in which distinct paralysis was present, degeneration of the spinal cord existed, although the disease otherwise bears a predominantly humoral character.

As regards the communication of the poison of rabies to man, it is consoling to think that not all who are bitten become affected, but that in a hundred cases of bites, the poison takes effect about forty seven times only. The wound itself generally heals readily; more rarely it suppurates for some time, which is regarded as more favorable; the local reaction is never so great as to threaten danger from thence, and in this respect the poison of rabies differs very essentially from the other animal poisons hitherto spoken of; it is not a phlogogenic poison. The outbreak of the disease seldom occurs before the sixth week after the bite, frequently later; a case

has been reported very recently in which the disease did not appear until after six months. Older writers give a much longer period of incubation. There is a rather general popular notion that the number nine plays a part therein, that the disease shows itself on the ninth day, in the ninth week, or in the ninth month after the bite, and that a person bitten is never quite safe until after the end of the ninth year. This is no doubt to be regarded as a fable easily explainable by the long incubation period, which has probably given rise to the idea. *Where* the poison remains during the long period, whether in the cicatrix, the nearest lymphatic glands, or the blood, is entirely unknown. It has been observed in a few cases only that the patient had pain and noticed a slight redness of the cicatrix a short time before the outbreak of the disease; then follow, first, great irritability, excitement, and restlessness, and in a few cases only, as early as this, twitchings on swallowing. The irritability becomes greater and greater; the light, every noise, every draught of air tortures these unfortunate patients, and may cause general convulsions and the most painful contractions of the throat. Now comes on gradually the actual hydrophobia, which is entirely wanting in dogs; the patients have intense thirst, but the moment they see any fluid, they are seized by horrible anxiety and convulsions; accessions of deep, convulsive inspiration sometimes follow, sleep is entirely lost, the patients are in a state of constant anxiety on the slightest noise, because all these things immediately excite the painful contractions, which ultimately extend to the whole body, and then lead also to regular accessions of fury, with a look of the most frightful anxiety. On the whole, however, these unfortunate ones are easily quieted by being spoken to gently, and are either completely resigned or very melancholy. They sometimes warn those about them not to come too near lest they should bite them, but are not at all malicious. Towards the end only, profuse salivation occurs, with foaming at the mouth. In some cases, death ensues after the most violent tonic convulsions; in others, extremely quietly, after the convulsions and aversion to fluids has ceased entirely, and both patient and doctor have indulged in a deceptive hope. Pathological anatomy furnishes us, unfortunately, with no explanation of this remarkable and frightful disease. There can be no doubt that the spinal cord is affected in it; whether the nerve-substance itself is diseased it has, as yet, been impossible to ascertain.

As regards the prognosis, we are, unfortunately, compelled to admit that for those patients in whom the disease has come to an outbreak there is no salvation. It might be advisable in all cases to cauterise or burn out deeply the bites of all rabid animals, and to keep them for a long time in a state of suppuration. This is, at least, the only thing which we can rationally undertake; whether the excision of such a cicatrix could still be of use if the disease has already shown itself cannot be determined from the investigations hitherto made; it might at all events be tried. Almost all the powerful medical and surgical remedies have been exhausted for the fully developed disease; all the narcotics have been administered in small and large doses; belladonna and opium especially have been given in almost poisonous doses, and by the artificial narcotism mitigated the sufferings of the patients even if no further good was effected. The cicatrix and the whole extremity have been amputated together, but in vain! Dieffenbach performed transfusion in such a patient, but in vain! In developed hydrophobia, we may administer fluids through a tube. The patients are most comfortable with absolute rest in a half-darkened room; for mitigating the convulsive attacks, repeated narcosis with chloroform has proved the most effectual remedy, and the patients who have once become familiar therewith always ask to have it repeated. This includes the small amount of help which we can render to these unfortunate people.

Unfortunately, in spite of the most rigorous sanitary laws in civilised countries, the number of people who die annually of rabies is still considerable. From statistical calculations extending over twelve to eighteen years, the annual mortality from hydrophobia in Prussia amounts to 71, in Austria to 58, in France to 24, and in Bavaria to 17.

The diseases last mentioned come so much within the province of the veterinary surgeon, of sanitary police, and of internal medicine, that I could give you only a short sketch of them here. You will find more exact information concerning them in Virchow's 'Special Pathology,' vol. ii, class Zoonoses, where also the special literature is given. Also in the 'Treatise on Surgery,' published by v. Pitha and myself, you will find in vol. i, section ii, detailed descriptions of the zoonoses. I also call your attention very especially to the article "Zoonoses" quite recently written by Bollinger in v. Ziemssen's 'Handbook of Special Pathology and Therapeutics.'

LECTURE XXVIII.

CHAPTER XIV.

ON CHRONIC INFLAMMATION, ESPECIALLY OF THE SOFT PARTS.

ANATOMY: 1. *Thickening, hypertrophy.* 2. *Hypersecretion.* 3. *Suppuration, indolent abscesses, congestive abscesses, fistulæ, ulceration. Consequences of chronic inflammations.* GENERAL SYMPTOMATOLOGY. COURSE OF THE DISEASE.

GENTLEMEN,—After having occupied ourselves hitherto almost exclusively with acute processes, we now come to those of a chronic character and, first of all, to chronic inflammation. I shall, however, adopt a different course here to that previously followed, inasmuch as I shall not enter at once into the individual forms of chronic inflammation occurring chiefly in surgical practice, but shall give you first of all a general description of the process itself.

In chronic inflammation, as well as in the acute form, it is a question of chemical and morphological changes in the tissues and of derangements of nutrition in them; these are followed partly by softening and breaking up, partly by molecular decay, or more extensive, slowly produced necrosis of the tissues. Upon these processes supervene dilatation of vessels, exudation, and new formation of tissue. This combination of processes may assume very manifold forms; chronic inflammation leads to very complicated appearances, according as this or that stage of the process is more or less enduring, according as decay, softening, or induration of the affected tissue sets in, and according to the equally varying fate of the inflammatory new growth. In an ætiological point of view also, the circumstances in chronic inflammation are much more complicated, for it is there a question chiefly, not of a stimulus acting

once, not always of a simple injury, a burn, a contusion with its consequences running a typical course, but (1) of the explanation why the respective inflammation, concerning the direct causes of which we frequently learn nothing from the patient, sets in at all, and (2) why it assumes a chronic character.

I will explain to you, first of all, what *anatomical changes* takes place in the tissues in the processes of chronic inflammation, in doing which I shall, here also, start chiefly from the connective tissue as the usual seat of the disease, as in the case of acute inflammation. Side by side with the dilatation and increase by the formation of loops of the capillary vessels, we have, in acute inflammation, become acquainted with the serous and plastic infiltration of the tissues as the most essential anatomical appearances. In chronic inflammation, the dilatation and increase of the capillary vessels are less prominent, while the morphological changes of the tissues, especially by the infiltration of new growth in them, and the serous infiltration are destined to play a greater part. The cellular infiltration of the tissues takes place as in acute inflammation, but the individual cells often attain a somewhat more perfect development. The fibres of the connective tissue lose at the same time their tough, fibrous character; the subcutaneous cellular tissue loses its elasticity, and the consequence of this is that the tissue becomes visibly swelled, and appears gelatino-lardaceous and less movable than in the normal state. This is the commencing stage of every case of chronic inflammation. The course may vary as follows:

1. The tissues remain constantly at this stage of serous and partly plastic infiltration; the skin and subcutaneous cellular tissue, the capsules of the joints, the tendons, ligaments, fasciæ, in short, all those component parts of the body consisting of connective tissue which are in the condition here described, present on section transversely a pretty homogeneous, lardaceous appearance. We observe this most frequently in diseases of the joints and their neighbourhood, and since this swelling of joints occurs without any redness of the skin, it formerly received the name of *tumor albus*, a name which conveys no idea, it is true, of the nature of the process, but which, when confined to certain forms of joint disease, is practically useful. You can easily imagine that the tissue, as yet but little changed on the whole, may return from this diseased condition almost entirely to the normal state. The infiltrated serum

becomes absorbed, the newly formed cells become partly converted into corpuscles of connective tissue, and partly break up and die; the connective tissue itself returns to its former condition, or very nearly so. A state of cicatricial thickening continues for some time; in the course of time also, during the development of the chronic inflammatory processes, small extravasations may have occurred in the tissues in consequence of increased pressure upon the walls of the vessels. These become converted into a brownish-red pigment which, if present in large quantity, imparts a yellowish or grey colour to the tissue previously diseased. If no retrograde formation occurs, and the process goes on in the same form, the elements of tissue become more copious and thicker, and the whole tissue more massive under the influence of the constant excess of the materials of nutrition furnished to the diseased parts in consequence of the delay in the passage of the blood. Out of the infiltrated new cells fresh conective tissue is formed between the layers of the old, so that the skin, for instance, becomes in this manner three or four times as thick as before; this deposit of fresh similar tissue into the old tissue is called in pathological anatomy "*hyperplasia.*" If the thickening of the skin assumes a knotted form, it is generally called *pachydermatous*. Anomalies of secretion and changes in the formation of the epithelium then generally occur as consequent states, for in this form of disease of the cutis, the epidermis either becomes formed very copiously and rapidly becomes horny, or the horny change is incomplete and the epithelium of the skin does not become fully developed.

In the latter case, therefore, the inflammatory disturbance of nutrition does not cause any decay, any destruction of the tissue, but as it goes on itself in a slighter degree, so does it also keep up permanently the regenerative processes in a medium degree of activity, but permits only an incomplete formation of true tissue. Here lies the transition to the formation of tumours, to which subject we shall return later on.

2. If you represent to yourselves the process of chronic inflammation, so far as you are at present acquainted with it, transferred to a mucous or serous membrane, you will understand that, with the pathological changes which obtain in the tissues of those membranes, the secretion also cannot remain normal. There generally occurs an increase in it, a *hypersecretion;* chronic inflammation of a synovial membrane, for instance, or of a

mucous membrane, may show itself chiefly by such hypersecretion.

Chronic catarrhs of the mucous membranes may affect sometimes more the epithelial layers, sometimes those of the connective tissue, sometimes the glands of the mucous membrane; in many cases, all three suffer equally at the same time. In many cases also, the mucous membranes secrete, under such circumstances, almost pure pus, without themselves undergoing any considerable change. In these *chronic blenorrhœas*, the walls of the vessels are probably in such a state of permanent relaxation that they constantly admit of the passage through them of a great number of migratory cells. Somewhat different is the state of things with the synovial membranes of the joints. There are forms of chronic inflammation of the joints which manifest themselves by a very copious secretion of extremely watery synovia without any admixture of pus (dropsy of the joints, hydrops articulorum), others which consist rather in thickening of the synovial membrane with but slightly increased secretion.

3. Chronic inflammation may also run its course with *infiltration of pus* and *formation of abscesses*, and the more minute processes therein are the same as in acute inflammation, except that everything goes on more slowly. There occurs, for instance, in some part of the body a constantly increasing infiltration of the tissues with migratory cells, where the tissue into which these cells become infiltrated softens and undergoes molecular decay. The tissue next adjoining the first disease-nest gradually becomes similarly infiltrated and disposed to conversion into fluid cellular tissue of a purulent character; the infiltrated tissue is the more disposed to suppuration and decay if no considerable development of vessels takes place in it, and no qualitatively and quantitatively sufficient materials for nutrition are furnished to maintain the further development of the excessive accumulation of cells. In this manner an abscess gradually forms, a circumscribed suppurating cavity, the walls of which are constantly on the point of becoming converted into pus, of *suppurating away*. All this goes on gradually here and often without the symptoms usually present in cases of inflammation, often without pain, redness, or increased temperature of the affected part, generally also without fever. Abscesses of this kind, which form chronically, are therefore called *indolent abscesses;* for this chronic process of suppuration the

term "*ulceration*" is also employed. We might also very well speak of the whole suppurating cavity thus produced as a "*cavernous ulcer;*" general custom, however, has reserved this expression chiefly for small cavities of this kind, while the larger, slowly formed suppurating cavities are called indolent abscesses. If you examine the pus from such an abscess with the microscope, you will find that it is very rich in fine molecules, but rather poor in fully developed pus cells. This results from the circumstance that the pus had been confined for a long time in the body and has become modified, on the one hand, by the breaking up of the pus cells into molecules, on the other, by chemical processes of transformation. By means of the latter, especially, copious fatty secretions take place, particularly of cholesterine in a crystalline form. The appearance of the pus also to the naked eye has been changed by these metamorphoses, since such pus appears thinner and more transparent than in acute processes, and contains, perhaps, flakes of fibrin and shreds of necrosed tissue. An indolent abscess sometimes requires many months, or even years, before the suppuration of its walls from within outwards becomes so far advanced that the skin is pierced. In many cases, it happens even that such an abscess increases but very little in size in several years, that the process of ulceration in its walls comes to a standstill eventually, and that the latter become converted into a cicatricial capsule and the pus thereby completely encysted. When we have an opportunity of examining such abscesses, we find in them a fluid resembling an emulsion, sometimes with crystalline fat and sometimes without a trace of pus cells, so that it would be difficult to infer from the anatomical contents that the sac before us had been an abscess if the whole course of the disease did not prove it. Much more rarely does it happen that in the course of time, if the abscess ceases to enlarge, absorption of the fluid takes place and a caseous pulp is left behind.

If the abscess has burst externally, the pus escapes and, under favorable circumstances to be described more minutely presently, recovery may take place. To render this possible, however, the process of ulceration on the inner wall of the suppurating cavity must first cease, which only happens, in general, if a sufficient development of vessels occurs in the walls of the abscess; under the influence of these, the internal surface of the abscess becomes converted into a vigorous granulating tissue and there takes place,

then, partly a thickening and shrinking of the latter to form cicatricial tissue, partly a growing together of the opposed walls of the cavity, as in the healing of acute abscesses. Less and less pus comes away from the opened cavity, which eventually heals up altogether. For some time afterwards, the subcutaneous cicatrix of the abscess may be felt as a callous thickening; in the course of time, however, this also disappears and the cicatrix resumes the character of ordinary connective tissue. I will make you acquainted here at once with the technical name employed for such abscesses as have not been formed originally at the spot at which they come under observation, but which, partly by the sinking down of the pus, partly from the advance of the intensely active process of ulceration, chiefly in a particular direction, have undergone a change of place. Suppuration may occur, for instance, on the anterior surface of the vertebral column which, following the course of the loose cellular tissue behind the peritoneum and the sheath of psoas muscle, advances further and further downwards, and finally shows itself beneath Poupart's ligament in the form of an abscess. Such and similar abscesses are called *gravitating* or *congestive abscesses*.

The above-described healing up process does not always take place as quickly as could be desired, for the general and local conditions are, unfortunately, sometimes of such a character that after the escape of the pus, either a very acute inflammation with high fever is set up in the abscess, and pyæmia or febrile marasmus supervenes, or that the chronic process of ulceration extends slowly but continuously in the walls of the cavity in spite of the escape of the pus. In such cases, the openings of these large, often deeply seated cavities secrete continuously a thin, unhealthy pus. The openings of such cavernous ulcers are called *fistulæ*.

You may represent to yourselves the process of suppuration or ulceration just described, the chronic softening and decay of a tissue infiltrated with cells, as transferred to a surface or skin, and this would bring us to the *superficial* or *open ulcer*, but since this is a subject of especially great practical importance, a chapter must be devoted entirely to it later on.

4. Chronic inflammation may run another course very similar to suppuration, namely to *caseous degeneration of the inflammatory new formation*, *tyrosis* (from τῡρός, cheese, in the sense of new cheese or curd). Represent to yourselves, again, a great accumulation of young cells in the tissue, and further, that such a mass of cells

undergoes molecular decay at its centre without the supervention of exudation, and that a cheesy pulp is thus formed. The plastic infiltration advances slowly in the periphery of the caseous nest by the accumulation of migratory cells, while the infiltrated tissue rapidly undergoes the caseous metamorphosis, and thus the central nest becomes always larger and larger. Here, also, as in suppuration, the local cause of the decay is a deficiency of vascularisation keeping pace with the formation of cells, or even a rapid, complete destruction of the existing vessels; here, also, a process of ulceration is going on which may be termed dry or "cheesy ulceration" (*avascular, dry necrotisation*). When such yellow nests are found in the body after death, it is assumed by many that they always correspond to a dried up suppuration-nest; this is not the case, however, or in extremely rare cases only; the majority of these cheesy nests were from the first what they are at the time at which they are observed; they were never fluid pus. That these cheesy nests may be formed directly, without suppuration, from the inflammatory new growth may be shown very easily experimentally. If you set up a permanent process of inflammation in the subcutaneous cellular tissue of a rabbit, for instance, by the introduction of a foreign body (*e.g.* a skein of hair), there forms around the foreign body, in the course of a few days a yellow, caseous mass, which represents, indeed, for the rabbit the same thing as pus in the human subject, but has never previously been fluid pus. So there exist also diseased conditions in man in which, during the process of chronic inflammation, this caseous degeneration takes the place of suppuration.

The further destiny of these nests in man varies greatly. If the process takes place in a part lying not too far beneath the surface, it may, by advancing from within outwards, cause an external opening; the pulpy mass escapes and the cavity may close up gradually like an indolent abscess. It also sometimes happens that around old cheesy nests, frequently after months or even years, inflammation and suppuration set in and then the old contents become mixed with the fresh abscess-pus, and are discharged together with it. The course of things just described may be seen especially often in chronic inflammations of the *lymphatic glands*; the spontaneous discharge of the caseous nest there occurs very slowly, however, and such fistulæ of the lymphatic glands often remain at the same point for months and years.

Another termination is, that the caseous nest attains a slight development only, then shrinks completely and takes up such a quantity of salts of lime that eventually a *chalky* concretion is formed of it, which is surrounded by a cicatrix. This termination occurs, however, as already stated, in the case of small caseous nests only; it is frequent in the mesenteric glands, the glands of the hilus of the spleen, and the bronchial glands, very rare in all the other lymphatic glands of the body.

There is still another kind of chronic degeneration of certain organs which is connected with the deposition of a peculiar substance from the blood, the so-called *lardaceous matter* or *amyloid*, the relation of which to chronic inflammation is, no doubt, a more distant one. I shall not enter into this question here, because this form of disease occurs chiefly in the internal organs, and has, therefore, an indirect interest only for us.

As regards the *consequences of the chronic process of inflammation* first of all in a purely histological point of view, these are of various kinds. The cellular infiltration and the process of new growth take place principally in the connective tissue, and the result thereof is either a *restitutio ad integrum* or, after the destruction of the parts by the process of softening and ulceration, a cicatrix. If this process is set up in muscles or nerves, the tissues participate in a high degree secondarily. The contractile substance in the muscle as well as the medullary sheath of the nerve-fibres, often become destroyed therein from molecular decay, or fatty degeneration, in consequence of the disturbance of nutrition. Atrophy of the muscles and paralyses may, therefore, be the consequences of chronic inflammation. How far, under such circumstances, the capability of regeneration of the muscles and nerves extends cannot be determined; it appears, in general, to be very slight under these circumstances. Molecular decay and fatty degeneration may easily occur without inflammation of the connective tissue which surrounds the muscles and nerves. It does not, therefore, appear practical to me to designate fatty degeneration of the protoplasm alone as inflammation of the muscular tissue and nerves, as Virchow has done for the muscles at least. I should be more inclined to class these conditions with the various forms of atrophy, but with my present views concerning the theory of inflammation, I willingly admit that it is essentially a matter of

convenience only how far we shall extend the term "inflammation" in the field of chronic processes.

After these general anatomical observations, let us pass briefly in review the *symptoms of chronic inflammation.* They are the same as in acute inflammation, with the exception that they frequently occur in a different order and in different combinations, and generally show less intensity.

Swelling of the affected part is generally the first striking symptom; it depends partly upon serous, partly upon plastic infiltration; the parts feel doughy and more resistent than in the normal condition; if it runs on to the formation of an abscess, as may happen in the course of weeks or months, we find fluctuation, which gradually becomes more distinct. We shall only observe *redness* of the inflamed parts, since it is neither very intense nor extensive, on account of the sometimes slight dilatation of the vessels, if the parts lie on the surface of the body. A chronic inflammation of the mucous membrane of the nose, or of the conjunctiva, will be easily recognisable by swelling, redness, and an increase of secretion. In chronic inflammation of the skin, also, a bluish- or brownish-red colour will soon manifest itself; but if the inflamed parts lie deep, the skin is not at all changed in colour, and does not become red until the chronic inflammation finally extends towards the surface and implicates the skin, as, for instance, when an indolent abscess bursts externally. *Pain* is a symptom of chronic inflammation which presents the greatest varieties; in many very sluggish cases it is entirely absent, but may, under different circumstances, be very violent, and have a tearing, stabbing character, occurring sometimes rather spontaneously, sometimes more on pressure or even slight contact. Upon the pain and upon the anatomical changes which the parts undergo depends essentially the *disturbance of function,* which is, therefore, sometimes slight, sometimes considerable. Heat, a temperature which appears raised on applying the hand, is frequently not present at all, or in a very slight degree only, in the chronically inflamed parts.

Fever is by no means a necessary symptom of chronic inflammation; it is generally present only if the chronic inflammation assumes a somewhat more acute character, as not unfrequently happens in the course of the disease, especially if the body is

weakened in the highest degree by long-continued processes of suppuration. Then the so-called *hectic fever* sets in, a febris continua, or simple remittens, with very great differences in the morning and evening temperature of the body, a fever with steep curves. According to my view, this hectic, suppurative, or consumption-fever, results from the continued taking up of products of inflammation, especially of products of decay; it is, therefore, also most frequent and intense in rapid decay in the internal walls of large abscesses and in rapidly advancing processes of ulceration. These fevers often run their course with rapid emaciation, night sweats, and diarrhœa. Few individuals long support remitting, chronic suppuration-fevers. Thus I watched for a full year a boy of fourteen with a fistula remaining after resection of the head of the femur, and general lardaceous disease, during which time he had constant remittent fever; he succumbed later on with general dropsy.

The *course* of chronic inflammation may be brought, in general, under two rubrics; in the cases of the first, the commencement of the disease is very indistinctly marked, and can scarcely be given with certainty by the patient; in some instances it is a tumour, in others moderate pain, or a slight disturbance of function, which first called attention to a diseased condition. Cases which have begun unnoticed in this sluggish manner generally retain this character also in their further course. In other cases, the chronic inflammation is a residuum of an acute process; the chronic course is interrupted from time to time by acute accessions, accompanied by fever. Least of all can anything positive be said concerning the duration of chronic inflammation in general, since this depends, first of all, upon the causal elements, to which we shall return shortly. One thing, especially, I would ask you to bear in mind here, namely, *that the chronic processes of inflammation, like the acute, always contain in themselves the tendency to a termination*, to a typical end, since the new formation in chronic inflammation never proceeds beyond the development of altogether distinctly characterised metamorphoses of tissue, which, if the diseased tissue does not become broken up, lead, in one way or another, to the formation of connective and cicatricial tissue. Why it is important to bear this in mind will become clearer to you when we come to speak of chronic inflammation in connection with the circumscrip-

tion of other new growths, of tumours proper. That the chronic inflammatory new growth does not arrive at a typical termination unless the causes of it can be removed or cease of themselves, or if organs necessary for life are deranged, or the powers are exhausted by suppuration, is a matter of course.

LECTURE XXIX.

General Ætiology of Chronic Inflammation.—*Persistent External Stimuli. Causes of Disease existing within the Body. Empirical conception of Diathesis and Dyscrasia.*

General Symptomatology and Treatment of Unhealthy Diatheses and Dyscrasias.—1. *The Lymphatic Diathesis (Scrofulosis).* 2. *The Tubercular Dyscrasia (Tuberculosis).* 3. *The Arthritic Diathesis.* 4. *The Scorbutic Dyscrasia.* 5. *Syphilitic Dyscrasia.*

Local Treatment of Chronic Inflammation.—*Rest. Raised Position. Compression. Shampooing. Moist Warmth. Hydropathic Wrappings. Peat and Mud Baths. Animal Baths. Sand Baths. Absorbent Remedies. Antiphlogistica. Derivatives: fontanelle. Setons. Moxas. Actual Cautery.*

We come to-day to one of the most important parts not only of this section but of the whole science of medicine, namely, to the *causes of chronic inflammation.* We saw the acute inflammations arise after a stimulus, acting once only, and then run their course and terminate variously, but in a comparatively short time and typical manner, according to the anatomical conditions of the parts stimulated, and according to the nature and measure of the stimulus. We have now to do with processes of inflammation which go on for many months, often for many years. It must there be a question of a persistent cause, a constantly acting stimulus, or an abnormal reaction after simple stimuli.

Persistent stimuli may be of a purely local kind.—We will confine ourselves for the present to these. If small insects, such as itch acari, establish themselves in the skin by burrowing in the superficial layers of the cutis, lay eggs, and lead their busy life there, this is a persistent stimulus for the skin. This is followed by scratching,

and thus arises and goes on a chronic inflammation of the skin—the itch. If fungus-spores become deposited in the epidermis, and begin to grow there and increase to millions of minute vegetable formations, the skin is also brought into a state of continuous irritation by these obtrusive strangers; chronic eruptions ensue, such as favus, herpes tonsurans, pityriasis versicolor, &c. If persistent pressure or friction act upon the skin to a moderate extent, but continuously, this is also a chronic stimulus which generally occasions a thickening of the respective parts. The wheals upon our heels and many of our corns are the results of continuous friction and pressure occasioned by the modern coverings for the feet. In like manner, the workman who is occupied chiefly with hammer and axe has wheals on his hands, the shoemaker on the outward part of the little finger and side of the hand, &c. Further, foreign bodies situated in the tissues may keep up a constant chronic inflammation in their vicinity. Persistent or frequently repeated chemical influences upon the tissues may also cause chronic inflammation. Thus chronic catarrh of the stomach may be caused by frequently repeated drinking of schnaps or other strong liquors. Persistent obstruction of the blood or lymph, and coagulation of those fluids in the vessels, give rise, first of all, to hyperplastic processes in the walls of the vessels and in the immediate neighbourhood, to dilatation and the formation of loops in the collateral vessels, and sometimes also to diffused thickening of the tissues. The skin of the leg is especially liable to be thus affected if any permanent obstruction exists to the return of the venous blood from the extremity.

When it is a question of the removal of chronic inflammations which can be referred to such a persistent external stimulus, many more examples of which it would be easy to give, the result will be a favorable one. If we remove the animal or vegetable parasites, the persistent pressure, the chemical influences, &c., the chronic process of inflammation will, in most cases, cease of itself.

We have been speaking hitherto of the effect of a persistent local stimulus upon healthy tissue. If you represent to yourselves a rather powerful stimulus acting once only upon a tissue already diseased, you cannot expect that in such a case the same state of things will obtain as in a simple process of traumatic inflammation in a healthy tissue. It is more probable that the consequences of the stimulus acting once only will be different, and perhaps more enduring, because the conditions for the typical rectification of the

disturbance on the part of the tissue are no longer present as in healthy tissue. Take the case of a superficial contusion of a portion of skin already in a state of chronic inflammation; the occurrence of a chronic suppuration, or even of slowly spreading ulceration, may be the consequence of this stimulus acting once only, which, under normal conditions of the skin, would have led rapidly to a new formation of epidermis, and therewith to a cure.

Unfortunately it is possible in comparatively few cases only to discover such purely local causes for the commencement and continuance of a process of chronic inflammation. In a very large majority of cases the cause does not lie so near, and long and repeated observation was required before any connecting links for the ætiology of the majority of cases of chronic inflammation and of chronic diseases in general could be found. I have made no allusion here to miasms or contagions in connection with the ætiology of the processes of chronic inflammation, since there is nothing to show that brief exposure to such has any power to produce them. There exist, indeed, chronic miasmatic diseases, such as ague and others, but the deleterious influence is there persistent, and the disease can frequently only be cured by the removal of the patient out of the miasmatic atmosphere. This case, therefore, falls within the category of persistent external stimuli. The same holds good for taking cold repeatedly, since each fresh attack affects the body while still suffering from the previous ones, and thus leads to chronicity of the diseased condition. All this does not suffice for the ætiology of chronic inflammation. We therefore seek for the causes in certain states of debility, in congenital or acquired tendencies of individual organs, or of the whole organism. Let us now inquire what experience teaches us on this point.

The first thing which strikes us on careful observation is, that certain forms of chronic processes of inflammation occur over and over again in particular organs and at particular parts of the body, and also that these processes of inflammation present themselves predominantly at a certain age and in individuals who show a certain kind of external resemblance to each other. Thus we see individuals of a certain type who become affected especially with chronic swelling and suppuration of the lymphatic glands, the joints, or bones; others who are attacked rather by sluggish inflammations of the lungs; others who are disposed, in quite an extraordinary manner, to catch cold and suffer, sometimes at one point,

sometimes at another, from pain in the muscles and joints. We observe, further, that in the case of individuals of this kind, who always suffer again in an analogous manner, the special pathological characteristics are frequently transmitted to their posterity, they having themselves inherited them from their fathers or mothers, &c. To obtain a somewhat clearer view in this chaos of individual tendencies to disease, the persons disposed to certain chronic diseases were formed into groups: thus arose the thoroughly empirical division according to morbid tendencies or diatheses into lymphatic, scrofulous, tubercular, rheumatic subjects, &c., expressions by means of which it was sought first of all to point out, for instance, that scrofulous subjects are disposed to affections of the glands, tubercular subjects to the development of ulcerating modules, &c. This classification was carried further afterwards, and it was assumed that an entirely distinct morbid condition of the physiological processes in the whole body must underlie such a disposition to certain distinct forms of disease. A morbid material, a diseased entity, a materia peccans in the body was substituted. The blood and lymph presented themselves as the most convenient vehicles for this, since they pervade the whole body, and their condition furnishes, at least, a measure of the more or less normal or pathological state of the whole organism. The word *dyscrasia* signifies such a pathological condition of the blood; thus we speak of scrofulous, tubercular dyscrasia, &c. It is a strange thing, however, that all the burden of the pathological changes in the whole organism should be laid upon the blood alone, and that it should, to a certain extent, be assumed that an infection of the whole body results from it. This could only be admitted for cases in which an abnormal material is introduced directly into the blood from without, as you have seen, for instance, in the case of poisoned wounds. But this is not at all or only partly the case with the dyscrasias here in question, since the tendencies to disease develop themselves rather from little understood causes in the organism itself, if not already inherited from the parents. The blood is no more absolutely stable quantitatively or qualitatively than any other tissue of the body; it becomes renewed constantly, then partly used up, and again renewed, and so on. Where the source of the renewal of the biood corpuscles lies is not known with certainty. That the serum of the blood is constantly renewed from the lymph, and the latter chiefly by the chyle-vessels from the alimentary canal, and that from

the blood a quantity of dissolved matter, salts, extractives, gases, and water, is excreted by the kidneys, lungs, skin, &c., is known to you from the lectures on physiology. How little do we know comparatively of these things, and how complicated are these few relations! I turn your attention to this to point out to you that normal blood can only proceed from a normal body, and that we cannot speak at all, therefore, in a physiological sense of a partial diseased condition of the blood standing in no kind of relation to the tissues. It could serve no good purpose, however, to exclaim against the use of the words "dyscrasia," and "diathesis," so generally introduced into medical language, and to endeavour to banish them altogether. It will do no injury to science if we continue to use these expressions in the accepted sense. It is still convenient for the present to have a designation for these things, since the latter are not imaginary, but are founded upon observations verified during centuries, although the interpretation of them has become variously changed in form in the course of time. We may, however, go too far with the classification of individuals in this direction, namely, by attempting to establish for every man a pathological diathesis, or to place every patient in one of the chief classes. You must not assume that it is easy to place every patient, according to his individuality, in one of the classes formed, as we analyse and classify a plant, for, since all kinds of men are capable of procreation with each other, a number of intermediate forms naturally arise which resist all classification. That all the physical and psychical characteristics of man do not gradually converge to form a comparatively average type lies in the hereditary conditions of all organic beings, in accordance with which, under the given circumstances, the types always inevitably present themselves again. This law naturally holds good also for those characteristics and tendencies which we are compelled to designate as pathological. There are physicians, it is true, and have been in all ages, who deny altogether, with exaggerated scepticism, the existence of a general morbid tendency to certain forms of disease, and regard everywhere only local, or partly only accidental stimuli as causes of disease. Such a hypersceptical current of thought ran recently through the whole of modern medicine, and was fully justified when the crasis-theory became so rife that there was scarcely any process of inflammation, it may even be said scarcely any disease, for which a specific crasis was not substituted. Any one who observes carefully and impar-

tially, and has the opportunity of seeing many patients with various modifications of disease, will no doubt acquire correct views in time, and neither rush too recklessly into the crasis-theory, nor set aside as illusions and deceptions all the experiences collected on the subject in the course of centuries. It may be questioned whether it can serve any practical purpose to employ at all terms such as scrofulous, syphilitic, and other inflammations, or whether it would not be better to study the chronic-inflammatory processes without any reference to their origin. The future will settle this question. For the present, I consider it to be my duty as a teacher to render your views of these things as clear as possible, and to enable you to come to an understanding concerning them with all your colleagues, to whatever school they may belong. You will seldom hear me speak, indeed, in the clinique of scrofulosis, &c.; but enough has now been said concerning these questions in a general way, so I will give you a brief sketch of the individual diatheses and dyscrasias as they are accepted at present.

1. *The lymphatic or scrofulous diathesis, scrofulosis.*—This tendency to a particular form of disease prevails chiefly during childhood, but it is by no means unknown at later periods of life. We assume the existence of this diathesis in individuals, especially children, who are much disposed to chronic-inflammatory swellings of the lymphatic glands, even after slight irritations, to certain catarrhal inflammations of the external skin (eczema, impetigo), especially of the face and head, to catarrhal inflammations of the mucous membranes, especially of the conjunctiva, but more rarely of the intestinal canal and organs of respiration, and to chronic inflammations of the periosteum and the synovial membranes of the joints. As regards swelling of the lymphatic glands, and especially of the submaxillary and occipital glands, it has been asserted that it only occurs in consequence of an irritation, *e.g.* dentition, or of eczematous eruptions on the head, inflammations of the eyes, suppuration of the ear, &c. This is partly correct, but even if we take the view that all swellings of the lymphatic glands are secondary affections, the very circumstance that in consequence of dentition, for instance, the glands swell, shows an abnormal formative excitability of the lymphatic system which does not by any means exist in all children; moreover, such local irritations cannot always be demonstrated for the almost equally frequent affections of the bronchial and mesenteric glands. It is

also a morbid state of things that the swellings of the glands in such individuals outlast the irritations, and may even, apparently without fresh cause, increase later on. It may be admitted that many of the diseases mentioned, *e. g.* some of the diseases of the joints in children, are set up by a slight injury, a contusion, and the like, but that they run a chronic, and, in part, an entirely peculiar continuous course, depends upon abnormal conditions in the tissues, which abnormity extends to the whole body in such a manner that it must be regarded, not as a purely local, but as a universal one. Various attempts have been made to explain this local and universal abnormity, and especially to refer the cause of the assumption of a chronic character to the continuance of the irritation, for the purpose of escaping from the mystery of one organism reacting differently upon a given stimulus to another. It has therefore been assumed that the substances which became formed in some way in the organs through a chemical change in the tissues are not taken up by the lymphatics and blood-vessels, and are not removed from the diseased organs, but remain in them and furnish the continuous stimulus to inflammation. I am far from wishing to deny that this may sometimes be the case; but admitting that it is equally correct for all cases, the peculiarity of certain organs just mentioned always remains an abnormity in those individuals, or a peculiarity of the forms of inflammation occurring in them under strictly defined circumstances. In short, we cannot get any further in this way than that we have to do with persons who, whether as regards certain tissues or in toto, differ from the majority of the human race. Children fall times without number upon their knees, hips, elbows, &c., generally without any evil results, or with such that the contusion disappears in a few days, even if no treatment was employed, and even if the contusion was a considerable one, as shown by the extensive extravasions and great amount of swelling and pain. Some children, however, have chronic inflammations of the joints even after slight contusions. These are exceptions, but no objection can well be made if we regard them as a special pathological class of human beings, and give a name to this class, thus seeking to classify them according to their natural peculiarities.

The attempt has been made to diagnose the scrofulous diathesis from the whole appearance and demeanour, the habitus of children. The following sketch is generally regarded as typical for a scrofulous

child. Light hair, blue eyes, a very white skin with considerable development of adipose tissue, thick lips, a large abdomen, voracious appetite, and tendency to constipation (torpid scrofula). You will meet with many resemblances to this portrait in practice, but also many children who do not resemble it at all, and yet suffer greatly from various forms of chronic inflammation. I do not attach much importance, on the whole, to these outward appearances. As regards the course and termination of the processes of chronic inflammation which occur in scrofulous children, the following may be said. In a few cases the chronic inflammatory swelling runs a retrograde course after a longer or shorter time, to complete *restitutio ad integrum.* Most frequently the course is accompanied by suppuration, which may, according to the various nature of the cases, assume a rather acute character, as happens with inflammation of the submaxillary lymphatic glands, and in diseases of the joints. The process very frequently retains for years a chronic character: abscesses, fistulæ, ulcers, &c., form. Early suppuration occurs, especially in somewhat emaciated, weakly, ill-nourished children, who readily become feverish (*erethetic scrofula*), in which cases the prognosis is particularly unfavorable. In many organs, in the lymphatic glands and lungs for instance, the *termination of chronic inflammation in caseous degeneration is very frequent;* the most deleterious influence upon the entire nutrition must naturally result if the mesenteric glands become degenerated in this manner, and the chyle-vessels thereby become in a great measure obstructed. An incurable atrophy of the whole body may be the consequence thereof.

The lymphatic diathesis is, in most cases, congenital and inherited from generation to generation. It may, however, be acquired by an injudicious mode of life, and, under improved circumstances, disappear to return once more, and so on. The following are regarded as chief deleterious agents in its production: living chiefly or exclusively upon potatoes, flour, bread; damp, unhealthy dwellings; want of cleanliness, fresh air, &c. It is very difficult to prove whether all this is correct. At all events, scrofulosis would prevail much more extensively amongst the poor than it does in reality if the conditions just mentioned *always* set up the scrofulous diathesis.

To sum up in a few words what is usually understood at the present day by lymphatic constitution or scrofulosis, it may be described—(1) *as a disposition to chronic inflammation of the mem-*

branes, bones, and joints, in which the inflammatory process may lead to the development of granulations, suppuration, and caseous degeneration; (2) *individuals are very commonly called lymphatic or scrofulous in whom swellings of the lymphatic glands, even if resulting from temporary irritations, run on for a long time, or even increase independently of any fresh peripheric stimulus.*

We will now speak of the *treatment* of scrofulosis in general. Above all things it is necessary to regulate the diet; good butcher's meat, eggs and milk, good well-baked white bread, baths from time to time, residence in fresh, healthy air, and an invigorating mode of education are the most important but often least available means to this end. In making the dietetic arrangements, regard must be had to the individual case, especially as to whether there is a tendency to obesity or atrophy, and whether the digestive organs are in a normal condition, or have been ruined by injudicious food from youth up. Since the disease is very common among the poor, although not altogether confined to them, these dietetic and hygienic remedies are very difficult of application. The number of internal remedies employed against scrofulosis is very great, but it is no longer a question, as formerly, of introducing a specific remedy into the blood as antidote for an unknown poison circulating in it, for such a poison does not exist, but of a purely symptomatic, for the most part general treatment. You see from this that "scrofula" is not a materia peccans circulating in the blood, but only a weakness of the organisation in a certain direction, a sometimes more, sometimes less, intense tendency to particular forms of disease. This is an essential difference, an essential advance beyond the earlier conceptions of the disease. After my explanation, you would be able to come to an understanding with those modern sceptics who are of opinion that all processes of chronic inflammation in children have a similar origin, and that it is, therefore, quite unnecessary, in every case of chronic swelling of lymphatic glands, or of chronic inflammation of the joints, to add that it is of scrofulous origin, or depends upon a lymphatic diathesis. It is possible that these expressions may disappear in the course of time, because they are useless for rendering our views more clear; it is not true, however, that all chronic-inflammatory processes in children are of similar origin, for it may, for instance, be a question of hereditary or acquired syphilis, and in adults, various other constitutional tendencies exist besides that which has hitherto been termed the

"scrofulo-tubercular," and which consists precisely in the tendency to chronic inflammations terminating in suppuration, caseous degeneration, and ulceration. That these processes stand in a certain opposition to other chronic forms of inflammation, *e.g.* to those which depend upon interstitial proliferation of the connective tissue (cirrhosis of the liver, Bright's disease, gray degeneration of the spinal cord, &c.), and lead to sclerosis, appears to me to admit of no doubt.

Many attempts have been made to improve the lymphatic diathesis: occasional purgatives were formerly employed, and in England especially, also mercury in small doses, which is very suitable for fatty, scrofulous children. Burnt sponge, the leaves of the juglandis regiæ, herba jaceæ, acorn coffee, and the bitter medicines were recommended, and are still very much used in the present day. In our time cod-liver oil is regarded as the best anti-scrofulous remedy, not only because it is supposed to counteract specifically the scrofulous diathesis, but because it is justly esteemed as a powerful aliment, and therefore strongly recommended for emaciated scrofulous children; for stout children it might eventually prove deleterious. Preparations of iodine are also much used in scrofulosis, but should be employed with caution and rather for stout than for atrophic children. Iodide of iron is more especially fitted for children who are at once stout and pale, and have fungous inflammations of the joints. The easily digestible preparations of iron are also very valuable remedies in cases of scrofula and anæmia. A good effect is obtained from mineral baths also, which may either be taken on the spot—in Germany, for instance, at Kreuznach, Rheme, Wittekind, Coblenz, Tölz, Reichenhall; in Austria, at Hall, Ischl, Aussee; in Switzerland, at Lavey, Rheinfelden, Schweizerhall, Bex—or they may be prepared artificially at home by adding from 16 to 50 ounces of salt to a tepid bath according to the size of the latter. For somewhat larger children sea baths are to be recommended; for weakly children warm baths, with the addition of malt and aromatic plants. For stout, scrofulous children, Niemeyer recommends hydropathic wrappings of the whole body, from which I have seen good results in some cases. Many physicians also recommend sulphur baths, especially the hot ones, for scrofulous affections of the joints; but the cases must be selected with great care, since the hot sulphur springs may also do harm in very painful, subacute affections of the joints with general

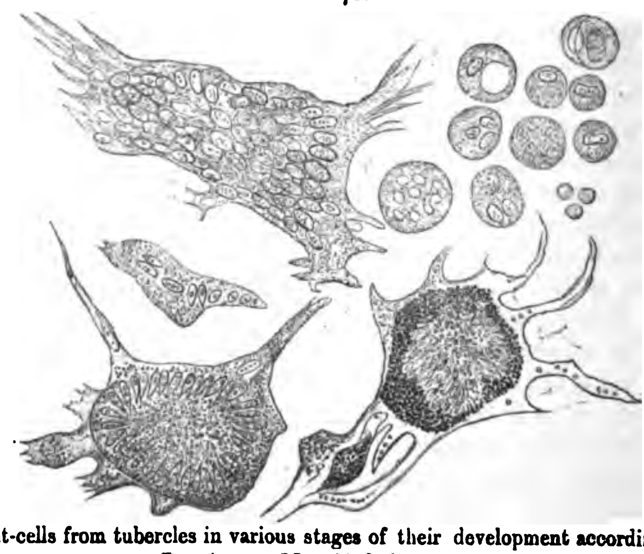
depression. You see that there is no deficiency of remedies, but we seldom succeed in improving the constitution by means of them, or in preventing the outbreak of fresh local processes in all cases. The local process also frequently goes on to such an extent that it becomes dangerous to life, and that local remedies must be chiefly relied on. In the course of years, the disposition to these affections decreases, as already stated, considerably, but many children die of scrofulous diseases of the bones and joints.

2. *The tubercular dyscrasia, tuberculosis.* The name of this disease is taken from tuberculum, a nodule, because the product of the disease presents itself in the form of very small, at first grey, afterwards yellowish-looking nodules, scarcely as big as a millet seed, or often microscopic, the so-called *tubercles.*

If you examine such a nodule with the microscope, you find that it consists of a mass of moderate-sized round cells, which are very distinct at the periphery of the nodule, while at the centre there is a fine molecular dry pulp, which, if the nodule becomes larger, assumes a yellow cheesy appearance.

The most recent investigations by Schüppel, Langhans, Rind-

FIG. 78.



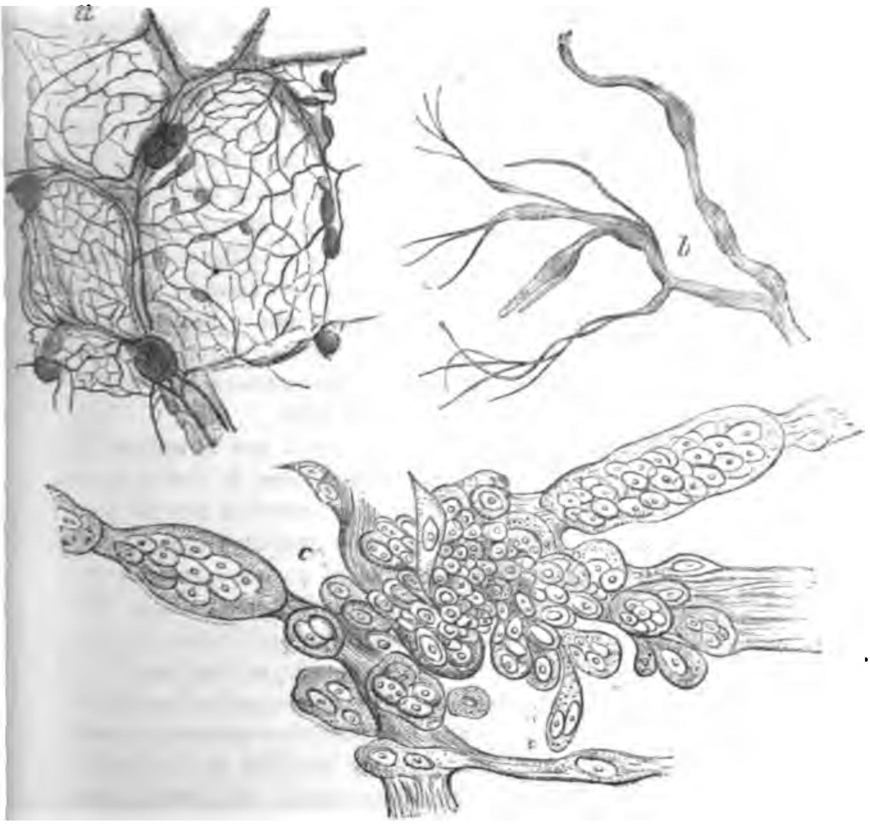
Giant-cells from tubercles in various stages of their development according to Langhans. Magnified about 400 times.

fleisch, and others, agree therein that at the centre of perfectly fresh

tubercles large masses of protoplasm, with numerous nuclei, so-called giant-cells, are found, such as we shall have to speak of later on, especially in connection with new formations in bone. The nuclei in the giant cells of tubercles very frequently have, however, a distinct peripheric arrangement.

The existence of these giant cells in tubercles is, however, not quite constant. We often see, in the peritoneum especially, a rather

FIG. 79.



a. Very minute tubercles in the peritoneum; *b*, very minute tubercles in an artery of the brain; *a* and *b* magnified microscopical preparations by Rindfleisch. *c*. Development of very minute tubercles in the peritoneum according to Kundrat. Magnified about 500 times.

irregular accumulation of large and small cells as the commencement

of the formation of tubercle, and in the neighbourhood of these, sometimes distinctly round, sometimes very irregularly shaped, but always sharply circumscribed new formations, are also found more diffused infiltrations (tubercular infiltrations), which are scarcely distinguishable from ordinary inflammatory infiltrations, except that the cells are about twice as large as the migratory cells which form the first cellular infiltration in acute inflammation.

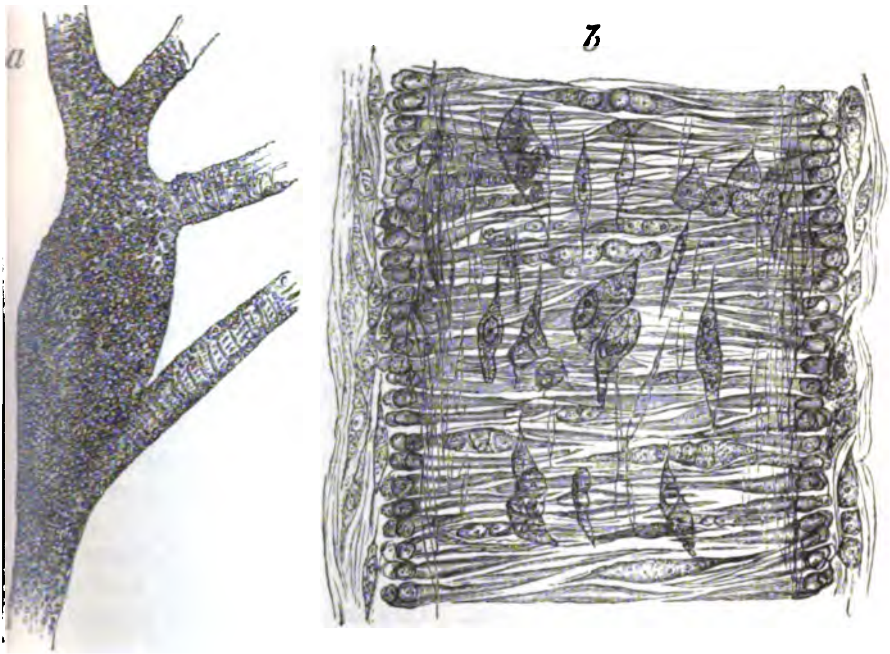
A very striking peculiarity of tubercles, especially pointed out by Rindfleisch, is, that they are often found in the walls of small and even very minute arteries, and in lymphatic vessels, but extremely rarely in veins.

Very various opinions may be held concerning the origin of the cells which form tubercles. If they are migratory cells they ought to increase rapidly in size after their egress from the capillaries and veins: on the whole, modern investigators incline but little to this view. Rindfleisch, Kundrat, and others, are of opinion that the tubercle cells arise chiefly from proliferation of epithelium, especially of the epithelium of the blood-vessels and their sheaths, and also of the lymphatic vessels and serous membranes. Rindfleisch also believes that tubercle cells may develop themselves out of the muscular cells of the arteries; Ziegler has demonstrated that they may also form by the confluence of migratory cells.

As regards the further fate of these small new formations, that which is most essential and peculiar about them is, that in general no vessels form and enter into them any more than into the purely epithelial new formations, although their periphery becomes freely supplied with vessels. Very rarely indeed do cases occur in which the tubercles gradually become converted into fibrous nodules. While every other new formation is connected with proliferation of vessels, this is entirely wanting, therefore, in tubercle, as has been insisted upon quite recently by Rindfleisch, Heitzmann, and others, and the consequence of this is that the new formation cannot long retain vitality; it dies at its centre, and only the cells at the periphery survive. The dead centre sometimes wastes, with fatty degeneration, to a pointed amorphous substance, which has the appearance to the naked eye of a dry cheesy mass; in short, the tubercle passes regularly, in consequence of its deficient vascularity, into a state of caseous degeneration. The enlargement of a tubercle might possibly go on *ad infinitum* from every fresh cellular infiltration of the tissue around the primary nest, but this seldom happens in reality. The

large caseous nests which are met with in the brain, the testicle, &c., have arisen, in most cases, from the confluence of numerous small nodules, a large number of which are also not unfrequently observed in the neighbourhood of large cheesy nodules.

FIG. 80.



a. Very minute tubercle from an artery in the brain. Magnified 100 times, *b*. First commencement of increase of cells in a very minute artery of the brain. Whether the cells with numerous nuclei are migratory cells, cells of connective tissue, endothelium, or muscle, or whether they have arisen from conversion of the inner coat into protoplasm, appears to me as yet not demonstrable. Magnified about 1000 times. Both drawings after preparations by Rindfleisch.

We thus come to the behaviour of the tissues to the tubercles sown to a certain extent in amongst them, in connection with which I must remark that the appearance of miliary tubercles in an organ, or in a part of an organ, usually occurs extensively. Subacute inflammation generally sets in around the tubercles, with copious cellular infiltration and a high degree of vascularity. This may lead to suppurative softening of the tissues, chronic formation of abscesses,

and ulcerative processes. A cavity is thus formed which contains pus, shreds of softened tissue, and tubercles in a state of caseous degeneration. But the inflammation around the tubercles may also participate in the caseous process, and a large cheesy nest be thus formed, which surrounds the primary tubercles; this may afterwards become softened by suppuration at the periphery, or firmly encysted and chalky. If tubercles form in mucous membranes—as so often happens in that of the larynx, intestines, ureters, bladder, and uterus—there set in, in addition to the tubercular infiltrations and ulcerations, suppurative catarrhs, and extensive desquamation of epithelium. In all these cases, it may happen, although unfortunately it very rarely does so, that the disease-nest, after having undergone one kind of metamorphosis or another, becomes encysted by means of a new formation of dense connective tissue, and then, after the contents have either been ejected or become chalky, the cyst shrivels up to a firm cicatrix; but in the case of serous membranes, and especially of the peritoneum, it very frequently happens that the inflammation caused by the formation of the tubercles in them leads at once to the formation of fresh connective tissue, and that not only each nodule rapidly becomes encysted, but that the intestines become so closely adherent to each other and to the wall of the abdomen, that it may be almost impossible after death to separate them.

As regards the *occurrence* of tubercles in the different organs of the body, not one is altogether exempt from them, but some are especially predisposed thereto. They are met with more frequently in the lungs, more particularly in the apices. A great number generally form at once. They become confluent, the walls of the bronchial tubes participate in the process and break down, and the cheesy, partly softened contents of the tubercles are coughed up. Blood-vessels also become softened by the process, burst, and give rise to pulmonary hæmorrhages. It is not our object here to go into details on this subject. You will hear enough of this terrible disease later on in the cliniques. After the lungs, the formation of tubercles occurs most frequently in the mucous membrane of the larynx, then in that of the urinary passages, the intestines, and even in the rectum, where these tubercular ulcers and abscesses acquire a surgical interest also. Tubercles also occur in the bones, particularly in the spongy bones, and especially in the calcaneus, the bodies of the vertebræ, and the epiphyses of the tibia. They

are rare, on the whole, in the synovial membranes of the joints. Although the lymphatic glands are often affected in tuberculosis, it is frequently difficult to demonstrate the existence of miliary tubercles in them; but Schüppel found them there also.

The views concerning the ætiology of tuberculosis have become much changed during the last few years. No doubt was entertained formerly that the formation of tubercle is partly an independent disease arising spontaneously, and that partly a tendency to it is inherited. A tubercular diathesis was spoken of as well as a scrofulous diathesis, and both were regarded as related to each other, but not identical. Laennec first propounded the view that the small, nodular new formations (gray miliary tubercles) are always the primary form of the disease, and that they lead, by their confluence and growth, to the destruction of the tissue attacked. The division of tubercles into gray miliary nodules and caseous nodules, the very remarkable acute miliary tuberculosis with its clinical resemblance to typhus, and the connection of the formation of tubercle with other and especially chronico-suppurative inflammations, and those which lead to caseous degeneration, always remained obscure in many respects, although Virchow had rendered the idea of tuberculosis more confined and precise; and thus, at least, not every caseous new inflammation was called tubercle. It was reserved for Buhl, on the ground of most careful investigations, to bring forward the notion that acute miliary tuberculosis is essentially the type of tubercular disease in general. He found it combined, in most cases, with caseous or suppurative inflammation-nests of longer standing. He ventured upon the at that time very bold assertion that it always arose from absorption of substances out of these nests, and the carriage of very minute particles into all the organs of the body. According to this view tuberculosis would be an infectious disease, a kind of nodular exanthem upon and in internal organs, dependent upon the taking up of a deleterious substance, especially out of old cheesy inflammation-nests, in the lymphatic glands, the lungs, the bones, &c., during which some of these particles probably cause a specific infection, as emboli in blood- and lymphatic vessels. The investigations of the last few years have shown, in fact, that very many points of disorganisation, in the lungs for instance, which had up to that time been attributed without hesitation to the presence of miliary tubercles, are thickened and partly softened caseous nests, which must be regarded as

results of a simple chronic-ulcerative inflammatory process, since no miliary tubercles are found in them, but only comparatively large-celled (tubercular) infiltrations. From this it appears as if, in pulmonary consumption also, the formation of true tubercles is to be regarded as something secondary, frequent, but by no means necessary. Niemeyer has done good service for the practical application of this new view, according to which *there would exist, indeed, a congenital diathesis for chronic inflammations leading to caseous degenerations, but not a direct tendency to the primary formation of tubercles.* This view has quite recently been essentially supported by the success of experiments made to cause tubercular disease in animals, especially guinea-pigs and rabbits. In these animals, namely, any, the slightest persistent irritation excites inflammation, with caseo-suppurative products, and then results from this nest a tubercular dyscrasia, which manifests itself in the production, partly of miliary tubercles, especially upon the serous membranes, and partly of yellowish nodules in the lungs, liver, spleen, &c., and causes death. These highly interesting experiments, which were first undertaken by Villemin, and repeated by Lebert and Wyss, Fox, Klebs, Cohnheim, Menzel, Waldenburg, and others, always with similar results, although interpreted variously, appear to me to prove, as I have always firmly believed, that tubercle is only a peculiar form of the inflammatory new growth; so that Buhl's view may be correct. It is very important, however, to bear clearly in mind that the inoculations just mentioned succeed in such animals only as have a certain disposition to caseous degeneration of all the products of inflammation, as I pointed out distinctly formerly for the rabbit. Rindfleisch very correctly observes that those animals become tubercular of themselves if an inflammation of an enduring character becomes developed in them. In dogs, for instance, the inoculation of tubercle does not succeed.

If we now fully acknowledge the great progress which the theory of tuberculosis has made in recent times, we must still not conceal from ourselves that the interesting relations which exist between many chronic surgical diseases and tuberculosis of internal organs, and especially of the lungs, are by no means fully explained. Even if the number of the cases in which, after chronic suppuration of joints and bones and caseous degeneration of lymphatic glands, tuberculosis of the lungs occurs, is considerable, it happens at least just as often that death takes place from exhaustion in individuals

with suppuration of joints and bones of many years duration, in whose bodies no trace of tubercle can be discovered. There are also definite conditions under which absorption of the cheesy masses either does not take place at all, or under which these masses, even if absorbed, do not produce tubercle. This would be in favour of the view that not only a disposition to caseous degeneration of the inflammation-nests, but also a disposition to dissemination and tuberculisation must exist, and that both these dispositions need not necessarily be so combined as they are in rabbits and guinea-pigs. Precisely in the circumstance that a caseous nest forms around a small point at which inoculation was performed, and that from this one caseous nest a diseased condition becomes developed and disseminated to the internal organs, lies something very peculiar to the animals in question, as well as to many human beings. It is just this peculiarity which is called the tubercular diathesis, but I will not conceal from you that there are many pathologists who merely admit the frequent coincidence of chronic caseous and suppuration-nests, and refer both to a cognate though unknown cause. All this cannot affect my belief, however, in the great importance of the recent investigations just spoken of, or prevent me from regarding them as one of the most satisfactory advances in modern pathology.

The *treatment* of tuberculosis appears to assume a peculiar and at first sight changed position, in consequence of the more modern ætiology of the disease. It has now become necessary to ask ourselves the following question: are there any remedies or procedures by means of which we can prevent any one who bears caseous pus about or in him from undergoing tubercular infection? The answer to this must for the present be decidedly a negative one; the mode of infection is so little known, that on that account already we cannot speak of preventing the process. The period also which lies between the development of the inflammation-nest formed primarily, and the subsequent tubercular infection is entirely beyond calculation. There appear to be cases, for instance, in which the formation of tubercles in the lungs gives rise to chronic bronchial catarrh and desquamative pneumonia; others in which the latter symptoms are the primary ones, and are followed closely by the formation of tubercles; others, again, in which many years intervene between the appearance of these two forms of disease. In short, the varieties of the processes are very great, but all this does not

furnish any direct indication for the treatment. As regards the hereditary character of the disease, to which such great importance is justly attached in tuberculosis, many relations have been rendered somewhat more intelligible by the modern theory, and many earlier experiences harmonise readily with the modern views. If true tubercles can be formed by self-infection, there can be no question of a direct inheritance of tuberculosis in the strict sense of the word, but only the disposition to chronic-inflammatory processes terminating in suppuration and caseous degeneration, &c., is hereditary, in other words, the scrofulous diathesis only, not the disposition to produce tubercles, is hereditary. We must bear this in mind provisionally; the experience of family physicians is in accordance with it on the whole, but we must not forget that such propositions have only a general value.

Tuberculosis may prove fatal (independently of incidental complications, such as diffuse meningitis, pulmonary hæmorrhages, pneumothorax, empyema, peritonitis from perforation of the intestines, pyæmia, &c.), partly by the extensive processes of suppuration and the comparatively rapidly increasing febrile marasmus, partly by amyloid degeneration of the internal organs which supervenes upon the suppurations, and partly, in the last place, by acute miliary tuberculosis, *i.e.* by an enormous eruption of tubercles in internal organs, accompanied by general poisoning of the organism, in which the patient falls into a kind of typhoid condition. In the earlier stages of the disease recovery may take place, but a tendency to relapses will be left behind.

If we sum up what can be said concerning the chief points in the *treatment of tuberculosis*, it will amount to something like this: we cannot prevent with certainty either the formation of tubercles or their dissemination. Distressing as this sounds, it must be added that careful management can do much to check the development of those processes which so frequently bring tuberculosis in their train. The careful, early general dietetic and local treatment of chronic affections of the bones and joints, even the well-timed amputation of limbs or resection of diseased bones, may prevent the eventual development of tubercles. In like manner, the most studied care in cases of catarrh of any kind and the earliest possible cure of them will be the most effectual means which we can adopt to prevent tubercular infection. No change is called for, therefore, in reference to the treatment of tuberculosis. All remedies, all baths and health

resorts, all directions as to their mode of life, &c., which we give to such patients, refer and always have referred:—(1) To the cure or mitigation of existing catarrhs or other primary diseases; (2) to improving the nutrition of the for the most part emaciated patients; (3) to avoiding everything which might excite inflammatory processes in these individuals, and render them feverish. I must leave it to the lecturers on clinical medicine to make you more familiar in detail with the important principles of the treatment of this so common and so terrible disease.

3. *Arthritis* or *gout* is a diseased tendency which does not declare itself, generally, until the thirtieth or even forty-fifth year, or later; it is classed very commonly with chronic rheumatism, but differs considerably therefrom. True gout is a disease very rare with us, and differs from rheumatism by occurring in accessions, often only once a year, and at fixed times, while the individuals affected are in good health in the interval. Gout is a disease of the rich and, as the earlier physicians, who suffered themselves therefrom, shrewdly added, of the cultivated class. It occurs chiefly in men who lead a comfortable easy life, and is not unfrequently transmitted to succeeding generations, but generally occurs at an advanced age. Harvey, Sydenham, Romberg, and many other celebrated physicians suffered from gout. The inflammations which occur in gout are confined chiefly to certain joints and the parts around them. The joint between the metatarsus and the first phalanx of the great toe is especially often attacked. This constitutes true *podagra*, or gout in the foot. Also the wrist and phalangeal joints may be affected, and then it is called *chiragra*, or gout in the hand. In these inflammations the skin in the neighbourhood of the joints is also involved; it becomes of a shining red colour, swelled, and very sensitive, as in erysipelas; in rare cases these processes may also lead to the formation of ulcers. Thickening of the arteries (atheroma), with its occasional consequences, cerebral apoplexy and senile gangrene, are not unfrequently met with in gouty subjects. Obesity and affections of the liver and kidneys may also accompany gout, and we frequently observe in the urine urates or oxalates, which have passed from the kidneys into the bladder in the form of a fine deposit, or collect to form larger calculi in the kidneys and bladder. The presence of a considerable quantity of urates has been demonstrated in the diseased joints and sheaths of the tendons, sometimes to such an extent as to form a white, granular covering to the surfaces and capsules of

the joints. An attack of gout is usually preceded for a longer or shorter time by a feeling of general discomfort, which begins to go off as soon as the inflammatory process becomes set up in some external part, generally in a joint. These inflammations last from a fortnight to six weeks, and then frequently cease, leaving behind them a permanent thickening of the joint; but in other cases, the limbs affected continue for many years unchanged. In many old gouty subjects, such hard concretions are met with in the neighbourhood of the joints and the sheaths of the tendons, also in the skin, *e.g.* about the ear. If these concretions burst, we can remove the masses of chalk and urates with a small spoon; the complete suppurating away and closure of such open and very painful concretions then requires months; active operative interference in such cases is utterly to be disapproved of. An ordinary attack of gout never ends in suppuration, but always in dispersion. On account of this ætiological relation of the abnormal deposit of urates to the affections of the joints, gout has also received the name of *arthritis urica*.

The treatment of the attack itself, the gouty inflammation of a joint, must be distinguished from the treatment of arthritis in general. The arthritic inflammation of the joints almost always runs a typical course, which cannot be altered essentially by therapeutic interference. The chief problem for medical science to solve is to mitigate the very severe pain by lowering the inflammation; the employment of ice would do good service, if not to be dreaded for certain reasons, namely, lest with the frequently existing atheromatous condition of the smaller arteries the continuous production of very low temperatures might cause gangrene. To the application of cold compresses, cold fomentations with Goulard water, weak solutions of nitrate of silver, or leeches, no great objection can be raised, but many gouty subjects prefer to have the inflamed joints smeared with some mild ointment, and enveloped in wadding. Profuse perspiration, brought about, for instance, by drinking large quantities of hot tea, and hydropathic wrappings, are said to shorten the attack in many cases. In the treatment of the arthritic diathesis, courses of mineral waters occupy the first place. The internal use of Carlsbad, Kissingen, Homburg, Vichy, and other saline springs, is beneficial, and the warm baths of Teplitz, Ragatz, Gastein, Wiesbaden, and Aix-la-Chapelle, are to be recommended for gouty subjects. We must be prepared, how-

ever, for an attack of acute gout when these warm baths are used.

4. The *scorbutic dyscrasia* manifests itself by a great fragility or softness of the capillary vessels and consequent subcutaneous hæmorrhages, which occur partly from lacerations of vessels, partly from diapedesis. The essential characteristic of this disease is assumed to be a state of dissolution of the blood, without its being possible to describe more exactly the kind of change in it which leads to the diseased condition of the vessels. The disease is almost exclusively endemic, and is of little surgical interest. I shall return to it in the next chapter when speaking of ulcers.

5. The *syphilitic dyscrasia.*—Although it is not my intention to include syphilis in these lectures, it is necessary to their completeness that I should make a few brief remarks concerning it. Syphilis has of course become established at some period in human beings, like the diatheses already spoken of; it now spreads, however, by inoculation alone. The person inoculated is, from the moment that the virus has taken effect, syphilitic, dyscrasic. When we speak of syphilitic affections in general, we usually include three forms of disease under the name:

(1.) *Gonorrhœa,* a blennorrhœa of the vagina, afterwards of the urethra, which may extend thence to the excretory ducts of the testicles and prostate, and give rise to gonorrhœal prostatitis and orchitis; condylomata often form where gonorrhœal pus stagnates.

(2.) *Soft chancre,* a circumscribed, ulcerative process, generally on the glans or prepuce, which shows itself a few days after infection, and frequently causes swelling of the inguinal glands, through the lymphatic vessels, with a great tendency to suppuration.

(3.) *The true syphilitic ulcer, indurated chancre. Lues.*—The inoculation is here followed at once by general infection, while the first and second forms remain comparatively local. On inoculation with the secretion of a true syphilitic ulcer, the whole organism immediately becomes infected. In about a fortnight, sometimes not until a month afterwards, an indurated nodule forms at the point of infection and becomes developed into an ulcer. Then occur a series of chronic-inflammatory processes in the most various organs, which are, at first, rather of a productive character, but afterwards soon lead to destruction of the infiltrated tissues, and assume a destructive, ulcerative character. The following symptoms may present themselves in syphilis:—Maculated, papular, desqua-

results of a simple chronic-ulcerative inflammatory process, since no miliary tubercles are found in them, but only comparatively large-celled (tubercular) infiltrations. From this it appears as if, in pulmonary consumption also, the formation of true tubercles is to be regarded as something secondary, frequent, but by no means necessary. Niemeyer has done good service for the practical application of this new view, according to which *there would exist, indeed, a congenital diathesis for chronic inflammations leading to caseous degenerations, but not a direct tendency to the primary formation of tubercles.* This view has quite recently been essentially supported by the success of experiments made to cause tubercular disease in animals, especially guinea-pigs and rabbits. In these animals, namely, any, the slightest persistent irritation excites inflammation, with caseo-suppurative products, and then results from this nest a tubercular dyscrasia, which manifests itself in the production, partly of miliary tubercles, especially upon the serous membranes, and partly of yellowish nodules in the lungs, liver, spleen, &c., and causes death. These highly interesting experiments, which were first undertaken by Villemin, and repeated by Lebert and Wyss, Fox, Klebs, Cohnheim, Menzel, Waldenburg, and others, always with similar results, although interpreted variously, appear to me to prove, as I have always firmly believed, that tubercle is only a peculiar form of the inflammatory new growth; so that Buhl's view may be correct. It is very important, however, to bear clearly in mind that the inoculations just mentioned succeed in such animals only as have a certain disposition to caseous degeneration of all the products of inflammation, as I pointed out distinctly formerly for the rabbit. Rindfleisch very correctly observes that those animals become tubercular of themselves if an inflammation of an enduring character becomes developed in them. In dogs, for instance, the inoculation of tubercle does not succeed.

If we now fully acknowledge the great progress which the theory of tuberculosis has made in recent times, we must still not conceal from ourselves that the interesting relations which exist between many chronic surgical diseases and tuberculosis of internal organs, and especially of the lungs, are by no means fully explained. Even if the number of the cases in which, after chronic suppuration of joints and bones and caseous degeneration of lymphatic glands, tuberculosis of the lungs occurs, is considerable, it happens at least just as often that death takes place from exhaustion in individuals

with suppuration of joints and bones of many years duration, in whose bodies no trace of tubercle can be discovered. There are also definite conditions under which absorption of the cheesy masses either does not take place at all, or under which these masses, even if absorbed, do not produce tubercle. This would be in favour of the view that not only a disposition to caseous degeneration of the inflammation-nests, but also a disposition to dissemination and tuberculisation must exist, and that both these dispositions need not necessarily be so combined as they are in rabbits and guinea-pigs. Precisely in the circumstance that a caseous nest forms around a small point at which inoculation was performed, and that from this one caseous nest a diseased condition becomes developed and disseminated to the internal organs, lies something very peculiar to the animals in question, as well as to many human beings. It is just this peculiarity which is called the tubercular diathesis, but I will not conceal from you that there are many pathologists who merely admit the frequent coincidence of chronic caseous and suppuration-nests, and refer both to a cognate though unknown cause. All this cannot affect my belief, however, in the great importance of the recent investigations just spoken of, or prevent me from regarding them as one of the most satisfactory advances in modern pathology.

The *treatment* of tuberculosis appears to assume a peculiar and at first sight changed position, in consequence of the more modern ætiology of the disease. It has now become necessary to ask ourselves the following question: are there any remedies or procedures by means of which we can prevent any one who bears caseous pus about or in him from undergoing tubercular infection? The answer to this must for the present be decidedly a negative one; the mode of infection is so little known, that on that account already we cannot speak of preventing the process. The period also which lies between the development of the inflammation-nest formed primarily, and the subsequent tubercular infection is entirely beyond calculation. There appear to be cases, for instance, in which the formation of tubercles in the lungs gives rise to chronic bronchial catarrh and desquamative pneumonia; others in which the latter symptoms are the primary ones, and are followed closely by the formation of tubercles; others, again, in which many years intervene between the appearance of these two forms of disease. In short, the varieties of the processes are very great, but all this does not

in many cases, but this treatment must be carried out with great energy and perseverance.

Moist warmth, in the form of poultice constantly renewed, is also very efficacious; further, *hydropathic wrappings*, which consist in dipping a cloth folded several times together into cold water, wringing it out, and then folding it round the affected part. This is then to be covered with an airtight layer of oiled silk or gutta percha, and the dressing to be renewed every two or three hours. The skin, which has at first been cooled down considerably, soon becomes very hot. The dressing is then renewed, and the cutaneous vessels are kept in constant activity by the change from cold to warmth, being thereby rendered especially capable of absorption. These wrappings are of very great use in many cases. The warm and hot natural *mud* and *peat baths* have sometimes a very beneficial effect for the rapid absorption of all torpid infiltrations, as well as for neuralgic affections in chronically inflamed parts. At Pystian, in Hungary, hot springs discharge themselves into the mud of small streams. The affected limbs are placed once or twice daily in vessels filled with the mud thus heated naturally. In many watering places these mud baths are now prepared artificially. Equally beneficial are the peat baths of Franzenbad and Marienbad; the moist peat strongly impregnated with the highly acid chalybeate water is heated and used as stated above of the mud baths. It is not known whether the soluble mineral salts aid in the results; these local baths perhaps act only as large poultices. Fomentations with the water of *thermal springs containing iodine* are also said to favour absorption. They generally soon cause eruptions on the skin, and may, therefore, be classed with the derivatives. The lower classes are also very fond of *animal baths*, which consist in placing the diseased extremity amongst the intestines of an animal just killed, and keeping it there until the carcase becomes cold; a special charm is sought for in the animal heat, of the good effect of which I have not been able to convince myself. Lastly, the hot sand baths formerly so much in vogue must be mentioned, but they have scarcely any advantage over moist warmth.

Absorbent remedies.—Amongst the dispersive applications, fomentations with *Goulard water*, *infusion of Arnica*, and *Chamomila*, &c., have a certain reputation, which they only deserve, however, as moist and warm wrappings, not on account of the additions to the water; they belong rather to the class of the indifferent domestic remedies.

It may be desirable to employ them, because many patients have no faith in water alone, and can only be persuaded to persist in the application of moist warmth if we order something from the druggists. *Mercurial ointment, mercurial plaster, iodide-of-potassium ointment*, and *tincture of iodine* are also absorbent remedies which are employed alternately in chronic inflammations. I am by no means inclined to deny that they have any effect in chronic inflammations, but too much should not be expected from them. Tincture of iodine has also been injected in recent times into the parenchyma of lymphatic glands in quantities of 5—10 drops, but with very unequal results. I pass over here a number of the so-called dispersive plasters; they have little value as such, but act partly as slight irritants to the skin, partly only as uniform coverings for defence against detrimental influences. I order such plasters in many cases to prevent the patients themselves from using anything hurtful; the plasters most in favour popularly are: emplastrum minii adustum (empl. noricum, fuscum), emplastrum oxycroceum, emplastrum saponatum (empl. saponato-camphoratum), emplastrum conii maculati (empl. cicutæ), emplastrum de Meliloto; long use only of mercurial plaster has a medicinal action. I will also mention *electricity* as a dispersive remedy; its effect does not appear to be considerable, but cases are given in which it was employed with advantage. Further investigations should therefore be made concerning it.

Antiphlogistic means proper.—Ice, leeches, cupping are rarely employed, and with but slight, transient result in chronic lingering inflammations, but are as important in all intercurrent acute attacks as in the acute primary processes of inflammation. Ice is employed continuously by some modern surgeons, especially by Esmarch, in perfectly chronic, torpid inflammations also, and the result highly spoken of. When it is possible to apply it with great care and persistence for months together, it sometimes acts beneficially for the absorption of chronic, inflammatory infiltrations, especially in affections of the joints and bones. I also have seen some strikingly favorable cases of this kind, while in others no good result was attained.

Derivatives.—These formerly played a great part in the treatment of chronic inflammation. They acquired this name from its being believed that they removed the inflammatory process from its first position to another less dangerous one. They are remedies by

means of which cutaneous inflammations varying greatly in degree can be produced, remedies which, according to the experiences of good observers, are said to have an excellent curative effect in many cases. Many attempts have been made, but hitherto in vain, to explain the mode of action of these so-called "derivatives." All that is suggested at present is that these remedies, when applied in the vicinity of a chronic inflammatory process going on in a joint or bone, draw the blood and humours outwards to the skin. In many cases of very torpid processes of inflammation running their course with little energy and low vascularity, derivatives certainly act rather in an opposite way, *i.e.* the new, acute inflammatory process set up in the immediate neighbourhood of the chronic one causes a stronger flow to these parts generally, and the chronic, torpid process is converted into more energetic activity. We will not trouble ourselves further here, however, to ascertain the way in which these remedies act physiologically. This has always been a very thankless effort. I very seldom employ this class of remedies, but since the milder ones are still often used by other surgeons, I give them here one after the other.

Nitrate of silver in a concentrated solution (about 5 to 30), mixed with some fatty material and rubbed upon the skin two or three times a day, produces a dark brown, silvery coloration of the skin and a slow scaling off of the epidermis. This is one of the mildest of the derivative remedies, and is especially applicable in diseases of the joints in susceptible children.

Tincture of iodine, especially the Tinct. Iod. fortior (5 parts of iodine dissolved in 35 parts of absolute alcohol with ether), if painted on the skin morning and evening, causes a rather sharp burning pain. If this is continued for two or three days, a blistering of the epidermis ensues, sometimes over the whole surface to which the remedy has been applied. *Blistering plasters* act more quickly; well prepared empl. cantharid. ordinarium is cut into pieces of the size of a franc or dollar and fixed upon the skin. In twenty-four hours a blister is formed under it, which is to be cut open and a piece of lint placed upon it, which dries on firmly and falls off after three or four days, in which time the detached horny layer of the epidermis has been reproduced from the rete Malpighii. This blistering plaster may either be applied once in a larger piece, or a small fresh piece may be applied daily. Lastly, a plaster may be used which contains a very small quantity of cantharides only, and

causes nothing more than a persistent redness. This is the "empl. canth. perpetuum," or "empl. euphorbii;" it is employed for several days or weeks successively. Although the occasional good effect of the above-named derivatives in chronic inflammation cannot be denied, I wish to point out to you that tincture of iodine and blisters are much more efficacious in subacute inflammations, and in the slight acute attacks which intervene in chronic inflammation, than in the entirely painless indolent forms.

The remedies which I have still to mention are such as are followed by long-continuing suppuration, which may be kept up at the will of the surgeon by artificial external irritants. The employment of them has decreased so much within the last ten years that the number of the surgeons who still use them is extremely small. I myself no longer have recourse to them at all.

Unguentum Tart. Stib. and *Oleum Crotonis.*—Both occasion, if applied repeatedly to the skin for some time—say from six to eight days, or more quickly if the skin be irritable—a pustular eruption, the breaking out of which is not unfrequently accompanied by very severe pain. As soon as these pustules become prominent, the application of the remedy is stopped, and the pustules are allowed to heal. Considerable cicatrices not unfrequently result from this treatment. The local effect of these remedies is a very unequal one—sometimes very violent, at others, again, very slight.

By *fonticulus*, or *fontanelle*, is understood a wound in the skin made intentionally and kept in a state of suppuration. This may be effected in very different ways. If you first apply an ordinary blister, for instance, then cut open the blister and dress the part deprived of its epidermis daily with unguent. canthar. or some other irritating ointment, you thereby occasion a permanent suppuration so long as you continue to apply the dressing in question. Another way of producing a fontanelle is that of making an incision through the whole thickness of the cutis, and placing in this wound, according to the size of the fontanelle required, a number of peas, which are to be fixed in the wound by means of adhesive plaster. The swelling peas, which are to be renewed daily, irritate the wound as foreign bodies, and thus a simple ulcer is kept up artificially. The simplest way of producing the fontanelle is always that by means of an incision; but the skin may be destroyed with any caustic, and the wound left when the scab falls off kept suppurating by the introduction of peas.

The *seton* consists of a narrow strip of linen, or of an ordinary cotton lamp wick, which is passed under the skin by means of a particular kind of needle. It is usually applied in the nape of the neck in the following manner: you form with the thumb and index finger of the left hand as high a fold of skin as possible, pierce it at its base with the armed seton needle, and draw the latter through. When the seton has been in position untouched for some days, and suppuration has set in, you draw it as far forward as is necessary for cutting off the portion impregnated with pus, and repeat this process every day. In the whole canal in which the seton lies, granulations form which suppurate freely. The seton may be worn for weeks or months consecutively, and removed when we no longer wish to keep up the suppuration.

Another method of causing permanent suppuration is that of producing an eschar on the skin by means of a red hot temperature and treating the granulating wound resulting therefrom for a longer or shorter time, according to the effect desired, with irritating dressing or the introduction of peas to prevent cicatrisation. There are two different ways of effecting this, either with the so-called *moxa*, or with a *red-hot iron*. I will not describe the various kinds of moxas here, as they are now very little used. The simplest method of producing an eschar on the skin is by means of some *caustic paste*, or the *ferrum candens*. With one of the instruments already mentioned in connection with the various means of arresting hæmorrhage, different degrees of burning may be effected, even to carbonization of the skin, to a varying extent, form, or depth, according as one extensive suppurating surface, or several smaller ones, may be desired.

Almost all classes of remedies have been greatly in vogue for a time, as various theories prevailed, and thus there was a time in which moxas, or a red-hot iron, or fontanelles were held up as universal remedies for every chronic disease. An individual had an issue formed in his arm to defend him from rheumatism, piles, tuberculosis, or cancer, with the idea that the pus thus produced would carry off all the peccant humours from the body. In like manner, courses of treatment carried out yearly at fixed times with purgatives, emetics, bleeding, &c., were in favour. Even now you will not unfrequently hear older practitioners assert that one or other of their patients was saved from a number of diseases by the application of a fontanelle. I will not take upon myself to attempt

to define the limits of possibility in therapeutics, for we are, as already remarked, far from being able to measure physiologically the effect of derivative remedies. We must, however, be suspicious of the effect of such means as are recommended as universal remedies for all possible diseases.

LECTURE XXX.

CHAPTER XV.

ON ULCERS.

Anatomy.—External characters of ulcers; form and extent; floor and secretion, edges, circumference.—Local treatment according to local condition of the ulcers; fungous, callous, ichorous, phagedænic, sinuous ulcers.—Ætiology of ulcers; permanent irritation, obstructions of the venous circulation.—Dyscrasic causes.

The theory of ulcers is naturally closely connected with that of chronic inflammation. What an ulcer is, whether a given wound-surface is to be regarded as such, are questions about which surgeons are generally agreed in practice. To give a short definition of an ulcer is, however, just as difficult as to define an object in any other province of medicine or the natural sciences. To give you an approximate idea of what an ulcer is, I will say to you, it is a wound-surface which has no tendency to heal. You see here already that every large granulating wound with exuberant granulations, which comes to a standstill in the process of healing, may be regarded as an ulcer, and in fact, Rust, who made the most detailed, though now but little used, nomenclature with regard to ulcers, designated a granulating wound as "ulcus simplex."

From my own observations and investigations, I must still adhere to the opinion that the formation of an ulcer has its starting-point, for the most part, in a process of chronic inflammation, and that in such a manner that the decay of the tissues only occurs when the tissues themselves have already undergone cellular infiltration in consequence of the inflammatory change. A simple decay of the tissues, resulting from insufficient nutrition, cannot well be called

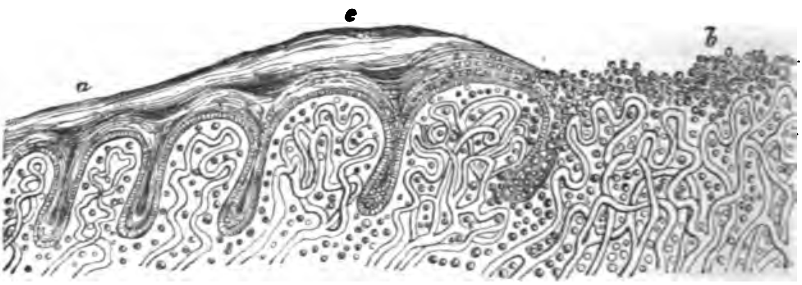
otherwise than necrosis. I admit, however, that here, as in all processes of inflammation generally, disturbances of nutrition, infiltration with migrating cells, and reproduction of tissue (sometimes more, sometimes less complete), are often so combined with each other that it is scarcely possible, in every individual case, to distinguish them strictly from each other.

The seat of a chronic inflammatory process leading to the formation of an ulcer may be in the deeper part of the cutis, the cellular tissue, the glands, the periosteum, or the bone. If suppuration, or caseous degeneration, or any other form of softening and decay, occur at the centre of such a nest, with gradual peripheric advance and perforation of the skin from within outwards, the so-called *cavernous ulcer* is formed; this, as already mentioned elsewhere, is an indolent abscess in miniature.

The seat of the chronic inflammatory process is frequently in the outer layers of the skin; here the *open ulcer* of the *skin* or *mucous membrane* is formed. We will make this clear by describing such a case. A chronic inflammatory process has been set up in the skin of the leg by one of the above-named causes, *e.g.* on the anterior surface of its lower third. The skin is traversed by dilated vessels, and therefore abnormally red, it is swollen partly from serous, partly from cellular infiltration, and somewhat painful on pressure. These are migrating cells, infiltrated chiefly in the superficial portions of the cutis, with more than the normal quantity of serum. The vessels increase in number and become dilated, the papillæ become larger and more succulent, there is increased development of the cells of the rete Malpighii, and its superficial layer scarcely attains the proper horny character, the connective tissue of the papillary layer is softer and has partly become almost horny. Slight friction now suffices to remove the soft, thin, horny layer of the epidermis at one point. The cellular layer of the rete Malpighii thus becomes exposed; fresh irritations supervene, and a suppurating surface is formed which consists of migrating cells in its upper, and of the already greatly degenerated and enlarged papillæ of the skin in its lower layer. If proper rest for the parts and defence against fresh irritation were secured at this stage, the epidermis would gradually form again, and the hitherto altogether superficial ulcer cicatrise over. But the slight superficial wound generally attracts too little notice, and fresh noxious influences of various kinds affect it; suppuration and molecular decay of the exposed, inflamed tissue, then

of the papillæ, follow, and thus a loss of substance, partly deeper, partly broader, gradually takes place. The ulcer is now completely formed. The following plate is the section of a spreading ulcer of the skin, from which the description of this process has been taken (Fig. 81).

FIG. 81.



Cutaneous ulcer of leg. Magnified 100 times; according to Förster. Atlas, Table XI.

You see at *a* the already somewhat thickened cutis, the papillæ of which increase in size towards *b*, while the loops of the vessels become larger, and the connective tissue is more and more occupied by cells; at *b* you see the fully developed surface of the ulcer, at *c* the greatly thickened epidermis, which forms the hard border of the ulcer.

You must represent to yourselves as exactly similar the process in the *mucous membranes;* first there is a more active migration of fresh cells towards the surface; this is soon followed by a moderate amount of serous and plastic infiltration in the connective tissue, with formation of fresh vessels in the mucous membrane and increased secretion from the mucous glands. As already observed, it was believed until lately that catarrhal pus is of a purely epithelial character; now it is rather thought that the elements of the catarrhal secretion consist chiefly of exuded white blood corpuscles. Permanent irritation of a mucous membrane affected with chronic catarrh is followed by softening and decay of the tissue, as was described above for the cutis; we then have before us a *catarrhal ulcer.*

There is another more acute mode of origin of ulcers, namely, from pustules which fail to heal, but spread after the pus has been discharged and maintain an acute, inflammatory character, *e. g.* soft chancres. Such ulcers also occur, especially without any recognisable specific, dyscrasic causes, from pustules of ecthyma on the

legs of young, often very full-blooded subjects who are quite healthy in other respects; they often assume an exuberant, fungous character, but in other cases they are accompanied by rapid decay of tissue. This more acute inflammatory origin of ulcers is much more rare than the chronic. Many affections bear half incorrectly the name of "ulcer;" thus, for instance, the "typhoid ulcer." In typhoid fever an acute progressive inflammation of Peyer's patches occurs, and this inflammation terminates in very many cases in gangrene, with necrosis of the inflamed portions of the mucous membrane; what remains after the throwing off of the eschar is a granulating surface, which generally heals rapidly. This granulating surface is not an ulcer in the stricter sense of the word, but only becomes one if the healing process does not go on normally; the "softening" (lentescirende) typhoid ulcer, in which the healing process is retarded, then first becomes a true ulcer. This by the way only; it is easy to employ these terms freely if we are quite clear about the processes themselves.

You see from this description that two kinds of process of an opposite character are combined with each other, as well in *ulceration* as in *inflammation*: new growth and decay. The latter occurs from softening of the tissues and suppuration, then also from molecular necrosis, or from both at the same time. The mutual relation in which new growth and decay here stand to each other admits of no doubt in the instances in question, for it is clear that in them the new growth preceded the decay. You might, however, represent to yourselves, as I have already pointed out, that in a portion of the skin previously healthy a disturbance of nutrition of such a nature may occur that a decay of the tissues takes place first, as you already know from the lecture on gangrene. At the edges of the sound, viable parts of the skin a new formation of fresh cells would develop itself, and if the parts in the neighbourhood of the primarily necrosed spot were healthy, the formation of a granulating surface and a cicatrix would then follow. If the parts are not healthy and viable to a slight degree only, fresh decay will occur in them also, instead of a vigorous inflammatory new growth, and an ulcer thus be formed and gradually spread. Such a case, in which an ulcer is formed primarily with molecular decay not preceded by cellular infiltration, occurs but seldom in practice. Molecular decay and gangrene are, indeed, strictly speaking, only quantitative varieties of the same process, namely, of the death of individual portions of

tissue. Cases may occur in which the ulcerative process and the gangrene stand extremely near to each other, as in hospital gangrene, of which we have already spoken, also, for instance, in the formation of the circular ulcers in the stomach, in which necrosis of the mucous membrane, in consequence of an extravasation, is usually the primary phenomenon. In most cases, however, the decay is always preceded by an inflammatory infiltration.

The observations just made, from which you see in what relation the process of ulceration stands, partly to the new growth, partly to the gangrene, will have shown you how difficult it is, in the field of these diseased processes, to distinguish everything with systematic sharpness. You need not fear, however, that I shall confuse you with further details here. We will proceed at once to the special characters of ulcers, where you will more quickly arrive at a clearer comprehension, and I will here only remark further that, as regards the vital process, ulcers may be divided generally into two chief groups, namely, into those in which the process of new growth predominates, which we will designate briefly *exuberant ulcers*, and into those in which the process of suppuration and decay rather takes the lead: these we call *atonic* or *indolent ulcers*. In between these two extreme limits of the anatomical and vital characters of ulcers lie a great number of varying forms. If the healing process is to be established in an ulcer, the first condition therefor is that the decay on the surface shall cease, then the floor of the ulcer must assume, approximately at least, the character of a healthy granulating surface, the cicatrisation of which occurs in the usual manner. In the case of indolent atonic ulcers, the development of numerous vessels and vigorous cells, which do not any longer lead to suppuration, but to a new formation of connective tissue, is absolutely necessary. In the case of exuberant ulcers, on the contrary, the new growth may be brought down to the normal degree. Herein lies, as you will easily recognise on further consideration, a suggestion as to the local treatment to be adopted in either case, of which we shall soon have to speak.

The *nomenclature* of ulcers varies greatly according to the characters which we especially look for in them. As in the case of chronic inflammation generally, we can distinguish two kinds, two chief groups of ulcers in connection with their origin, namely, the idiopathic and the symptomatic. The *idiopathic* (or *protopathic, proteropathic*) *ulcers* are such as result from purely local irritations;

we may also call them *irritation-ulcers*. The *symptomatic* (or *deuteropathic*) *ulcers* are such as present themselves as symptoms of a general diseased condition from internal dyscrasic causes, without any local irritation of the part affected.

Leaving aside for the present the ætiological conditions, we will first endeavour to render more clear, by the consideration of the external characters which an ulcer may present, in what the latter consists. When we attempt to describe an ulcer, we distinguish the following points in it:

1. *Form and extent of the ulcer.*—It may be circular, half-moon shaped, altogether irregular, ring-shaped, shallow or deep; it may form a canal leading inwards; it may be tubular, forming a *fistula*. These fistulæ arise, as I have already explained to you, from the formation of deep-seated inflammation-nests, either in the deep layer of the cutis, the subcutaneous cellular tissue, the muscles, the periosteum or bone, or also amongst glands, which gradually reach the surface by slow ulceration. The formation of a cavernous ulcer, a more or less deeply-seated suppuration-nest, therefore, always precedes the formation of fistulæ.

2. *Floor and secretion of the ulcer.*—The floor may be slightly sunk, deep, or projecting; it may be covered with dirty, stinking. serous, or ichorous fluid, or even with gangrenous shreds of tissue (*ichorous*, *gangrenous* ulcers). Its surface may also have an amorphous, a lardaceous, or a greasy appearance. The floor of the ulcer may also present too luxuriant granulations with spongy purulent secretion (*fungous* ulcers).

3. *The edges of the ulcer* are low or raised, vertical, hard (*callous* ulcers), soft, burrowing (*sinuous* ulcers), jagged, everted, undermined, &c.

4. *The circumference of the ulcer* may be normal or inflamed, œdematous, indurated, pigmented, &c.

These universally employed technical expressions suffice to describe exactly to any other medical man a given ulcer. But since the descriptions referring to the vitality of the ulcerative process, *i.e.* indolent, atonic, exuberant, fungous, &c., are in general shorter, we employ them more frequently. We also often employ descriptions which refer to the more remote causes, especially in the case of symptomatic ulcers. We then speak briefly of scrofulous, tubercular, syphilitic ulcers, &c. But while we have the local characters of an ulcer fresh in our memories, we will at once go through the

local remedies, in so far as their employment depends upon the nature of the ulcer itself. A great many ulcers, especially all those arising from repeated local irritation, heal very easily. So soon as the diseased parts are placed under more favorable external circumstances and no fresh injuries occur to them, cicatrisation often commences quite spontaneously. It is surprising how quickly the frequently occurring ulcers of the leg especially put on a much better appearance after the patient has had a warm bath, the ulcer simply been covered with a compress soaked in water, and the patient has lain quietly in bed for twenty-four hours. The ulcer, which previously looked dirty and of a greyish-green colour and smelt offensively, now appears quite different. Its surface now granulates tolerably, though not yet vigorously, and secretes healthy pus; a fortnight's rest and great cleanliness suffice in many cases to effect the complete cicatrisation of such ulcers. Scarcely has the patient been discharged, however, and returned to his usual occupations, when the cicatrix again gives way, and *in a few days* the state of things is the same as before. This goes on regularly, the patient comes back into the hospital and is soon discharged again, only to be readmitted in a short time. There are some means of defence against these relapses, however, of which we will speak later on. Not all ulcers tend to heal so quickly; many require various modes of treatment and much time for their cure. We will now go through the individual forms according to their local symptoms, in reference to the local remedies to be employed.

1. *Ulcers with inflamed circumference and congestive (erethische) ulcers.*—It very frequently happens that an ulcer, when first seen, if the patient is constantly going about with it, is very red and painful, and that this slight degree of inflammation subsides of itself after a certain time if the patient remains at rest. There are other ulcers, however, the circumference of which is always intensely red and painful, while the ulcer bleeds readily, and even the granulations are painful when touched. Such an ulcer is called an erethetic ulcer. The highest degrees of erethismus of the surface in ulcers are extremely rare. I had a patient in Zurich, who, in consequence of very intense phlegmonous inflammation of the thigh, had lost a large portion of the skin from gangrene; after the separation of the eschars, a very exuberant granulating surface was formed, which had little tendency to heal, and was so painful on the slightest touch that the patient cried out and shuddered. The cause of this

extreme sensibility in such cases has already been spoken of in connection with cicatrices of the nerves. As regards the treatment of inflamed and erethetic ulcers, we first try ointments of mild fat and wax, unguentum cereum, then so-called cooling ointments of zinc or lead, or fomentations with Liq. Plumbi Diacetatis. If the granulations continue painful and ill-looking with this treatment, we should try free cauterization of the surface of the ulcer with nitrate of silver, or still better, with the ferrum candens; the latter means, followed by compression with adhesive plaster, finally proved successful in the case mentioned above. The local employment of narcotics is usually recommended in such cases, especially poultices with an addition of opium, belladonna, henbane, and the like; but these remedies generally do so very little good that, in my opinion, we only lose time with them.

2. *Fungous ulcers, i.e.* those of which the granulations are fungous, exuberant, and project beyond the level of the skin. These ulcers secrete a mucous pus, and are especially rich in vessels.

Here we may employ astringent remedies, compresses with decoc-

FIG. 82.

Blood-vessels of two luxuriant points of granulation in an ordinary (not cancerous) ulcer of the leg, artificially injected by Thiersch. (Epithelial Cancer, Table XI, fig. 4.)

tion of cinchona or oak bark, but the good effect thereof is slight. It is best to destroy the surface of such granulations with caustics; daily use of the solid nitrate of silver suffices in most cases. When

this is not the case, we may employ caustic potash, or even the ferrum candens. Compression with adhesive plaster also often has a very good effect here. The simplest way is to cut off such granulations as often as necessary with scissors.

3. *Callous ulcers* are those most dreaded by the surgeon on account of the slow healing process in them. Their base, edges, and circumference have become thickened and of cartilaginous hardness from very long continued chronic inflammation. The ulcer, which is very indolent, generally lies deeply below the surface and has very sharp edges. Treatment must be directed to two points here, namely, to effect a softening of the tendinous edges so ill supplied with vessels and of the floor of the ulcer, and to bring about proper vascularisation as well in its edges as in its floor. There are ulcers of this kind which have existed for twenty years and more. The following are the means used for them:—Compression with strips of adhesive plaster, which are applied according to certain rules which you will learn in the clinique. Such a dressing with adhesive plaster, which must cover not only the ulcer but the whole leg, may be left on from one to two days at first, and afterwards, if the healing process has begun in the ulcer, from three to four days or longer. This so-called Baynton treatment of ulcers of the leg with adhesive plaster is of very great importance, especially for those cases in which the patients are not willing to remain at rest for a long time, being anxious to follow their occupations. I have had great experience of this mode of treatment of ulcers of the leg in the Berlin poly-clinique, but cannot give so favorable an opinion of it for them as many other surgeons do, who regard these strappings as a universal remedy for all such ulcers. In the case of out-patients, I consider these strappings to be very useful as coverings, since they serve to enable the patients to go about without causing the ulcer to spread too much, but that all ulcers heal especially readily under these dressings, and that the effects of the adhesive plaster upon the callous circumference of the ulcer are greater than those of the remedies to be mentioned further on, I cannot admit. The best means of keeping up a permanent congestion about the ulcer, and thereby facilitating the formation of blood-vessels and of tissue, is moist warmth, which you may employ either in the form of poultices or of a continuous water-bath, which is still better. The latter, by means of which an artificial swelling and softening of the

indurated circumference of the ulcer is produced, I especially recommend to you.

It is sometimes necessary to destroy the callous edges entirely, or to bring them to a state of high suppurative inflammation. The former can best be accomplished with the ferrum candens, the latter by the repeated application of the Ung. Tart. stib., or of the Empl. Cantharid. If the application of the latter remedy be followed by a pustular or even partly gangrenous inflammation of the ulcer and its circumference, you must place the leg in the water-bath and will effect a strikingly rapid cure in many cases. It is not always possible, however, to effect a cure of callous ulcers of the leg, and those especially which correspond to the anterior surface of the tibia and penetrate downwards as far as the periosteum are sometimes incurable. Such ulcers also as encircle the whole leg like a ring are generally classed with the incurable ones, and are regarded as an indication for amputation if they render the individual permanently incapable of walking—and especially of working.

Besides the conditions already mentioned, there is another circumstance which especially interferes with the healing of ulcers with a very indurated circumference, namely, that the healing, granulating surface and cicatrix cannot become smaller and denser in the usual way by strong contraction, because the firmness of the surrounding portions of skin does not admit of any movement. While every granulating wound, as you know, diminishes almost one half in size by contraction, and the surface of the cicatrix is consequently smaller, the granulating surface of these ulcers must, in many cases, cicatrise over the whole of their original extent, because they cannot contract. To render contraction possible, deep incisions have been made into the skin round about the ulcers, and kept open by means of lint, but I have not, as yet, seen much effect therefrom. Transplantations of epidermis on Keverdin's plan are made with advantage for accelerating the healing process, but it unfortunately often happens that these transplanted portions of skin, after they have healed well and the whole ulcer has cicatrised, again perish, and so the greater part of the advantage gained with difficulty is lost anew. Another consequence of the rigidity of the tissues is, that the not yet properly thickened fresh cicatrix very readily gives way, and the healed ulcer thus rapidly becomes established again. The best way of preventing this is to cover the cicatrix, when formed, with wadding, and to apply a starch bandage

to the leg. This dressing must be left on from six to eight weeks or more, until the cicatrix is definitively organised and firm. I have followed this plan for a long time with most ulcers of the leg after they have healed, and have reason to be content with it.

4. *Ichorous, gangrenous ulcers.*—The causes of processes of decomposition on the surface of an ulcer very frequently lie in the unfavorable external circumstances. In other cases, however, general dyscrasic conditions produce a tendency to a more rapid decay of the tissues on the surface of the ulcer. Solution of chloride of lime, pyroligneous acid, turpentine, spirit of camphor, carbolic acid, acetate of alumina, tar mixed with plaster of Paris, are the remedies to be employed here. If the decay of the tissues attain quite a striking degree of rapidity, so that the ulcer spreads considerably from day to day, the ulcer is called *eroding* or *phagedænic*, a form closely connected with what was formerly called hospital gangrene. The sprinkling of powdered red oxide of mercury rapidly arrests the decay in many cases. If this does not answer, I would advise you not to lose time about destroying the whole ulcer. Free cauterisation with caustic potash, or the energetic application of the ferrum candens, with destruction of the edges of the ulcer as far as the sound parts is, in these cases, almost always certain in its action.

5. *Sinuous* and *fistulous ulcers.*—Ulcers with undermined edges and fistulæ.—They always commence as cavernous ulcers, which gradually proceed from within outwards, and especially often from chronic suppuration of the lymphatic glands. Such an ulcer will generally heal most rapidly if you convert it into an open one by cutting away the usually thin, undermined edges, or, where this is not feasible on account of the too great thickness of the edges and walls of the cavity, by freely laying open, at least, the deep-seated ulcer. This treatment serves also for fistulous ulcers, if they lead to a more deeply-seated cavernous ulcer. The latter must first be healed before the fistula can become firmly closed. In the case of cavernous ulcers of the skin of the cheeks, and of suppuration of superficial lymphatic glands, which occur so often in the neck, I first cut the thin skin completely away, scrape off the floor of the ulcer with a sharp spoon, and apply lint steeped in Liq. Ferri Sesquichlor. Healing generally ensues rapidly and with less disfiguring cicatrices than if we trust to a natural cure, which may require months or years. I may remark here by the way that the word "fistula" has another meaning, since we designate thereby

every tubular, abnormal opening which leads to one of the cavities of the body; thus we speak of fistulæ of the chest, gall-bladder, intestines, bladder, urethra, &c.

We must now occupy ourselves with a very important part of the chapter on ulcers, namely, with their *ætiology*. I have already pointed out to you that we must distinguish here between local and general dyscrasic causes, as in chronic inflammation generally. All the causes of chronic inflammation would, therefore, have to be enumerated here; we will direct our attention more particularly to some of them only. If we first inquire more closely into the local causes of ulcers, the chief one will be found to be *continuous local mechanical or chemical irritation*. Continued friction and pressure are frequent causes of such irritation-ulcers. A tight boot, or the hard edge of a shoe, may produce ulcers on the feet; the growing of a toe-nail into the flesh is almost always the consequence of continued pressure by an ill-made boot; a sharp tooth, or sharp pieces of tartar on the teeth, may be the cause of ulcers of the mucous membrane of the mouth or tongue, &c. Ulcers of this kind generally bear the signs of irritation about them: the circumference is red and painful, as is also the ulcer itself. As chemical irritation I may mention the action of ardent spirits upon the mucous membrane of the stomach: drunkards suffer habitually from catarrh of the stomach, in the course of which ulcers of various kinds are not unfrequently formed.

A second, more frequent cause of chronic inflammatory processes terminating in ulceration are the already mentioned *obstructions*, especially in the *venous circulation*, and the pressure which these *varicose* distensions of the veins exert upon the surrounding tissues. These stand in very close relation to the origin of ulcers of the leg; we shall speak of them later on (Chapter XIX). I will merely mention here that, in consequence of the continued distension of the small cutaneous veins, a chronic serous infiltration of the skin is produced, which is followed gradually by cellular infiltration, thickening, and finally, pretty often also by suppuration and decay. The ulcers which result from varicose veins, and which are generally termed *varicose ulcers of the foot*, may present very different characters. At first they are usually simple, often exuberant ulcers, assuming only later on a more indolent character, while the edges at the same time become callous. I have already told you how quickly such ulcers become changed by rest and cleanliness. As regards

the treatment of them, the dressing with strips of adhesive plaster is very useful, as well to induce the healing of the ulcer as to prevent the further formation of varices. For the majority of cases, however, I prefer a treatment with a *raised position of the leg in bed*, according to the principles mentioned already, and only employ the plaster dressing later on to check the further development of varices.

If we have been led by our experience to bring varicose veins and ulcers into such close connection with each other, and thus pointed out the most important practical significance of this affection of the veins, you must not conclude therefrom that varices are always followed by the formation of ulcers; *a rather considerable number of cases occur, on the contrary, in which enormous varices exist unaccompanied by the formation of secondary ulcers.* That obstruction to the venous circulation does not, in itself, lead directly to inflammation has been stated already. But if, for months or years, a constantly increasing pressure from within bears upon the walls of the vessels, and the greatly distended, over-filled vessels themselves press upon the tissues, this pressure causes a slight inflammatory change, with chronic œdema and some interstitial formation of tissue (indurated œdema). Why this action is entirely absent in many cases I am unable to tell you.

We now come to a brief consideration of those forms of ulcer which result from *internal causes*, and are connected with dyscrasic states of the body, to *symptomatic ulcers*.

1. First in order here come *scrofulous ulcers*. These ulcers form with especial frequency in the neck, generally from within outwards, from the gradual formation of isolated suppuration-nests in the substance of the cutis or of the subcutaneous cellular tissue, which slowly break through the skin from within. Small losses of skin follow necessarily, the edges of which are generally somewhat red and very thin, and lead to more deeply-seated cavities which discharge tissues in a state of caseous degeneration, or thin pus. The edges of these cutaneous ulcers are undermined, as can easily be ascertained by examination with the probe. These ulcers are generally of an extremely atonic character. You see from this description that this form of undermined, sinuous ulcers depends alone upon their mode of origin, which may result from the most varied general constitutional conditions. Our experience teaches us, however, that the *great majority* of ulcers of this kind are met with

in scrofulous individuals, and for this reason the existence of scrofulosis is inferred from the occurrence of such atonic ulcers with undermined edges. This conclusion will be a correct one in most cases, although not always unconditionally.

2. *Lupous ulcers.* By lupus a disease is understood which announces itself by the development of small nodules in the superficial layer of the skin. These nodules may develop themselves further in various ways. They consist of accumulations of small round cells, with concomitant dilatation of the vessels, and mostly with softening of the infiltrated tissue. In many cases there appears to be present an exuberant formation of epithelium, advancing wedge-like in between the lupous nodules. The nodules may increase in size and become confluent, so as to form nodular thickening of the skin (Lupus hypertrophicus). On the surface, copious desquamation of the epidermis sometimes occurs (Lupus exfoliativus), or a process of ulceration (Lupus exulcerans). All these forms may be combined with each other, and there are some more which may be recognised.

The ulcers of Lupus exulcerans may be accompanied by the development of highly exuberant granulations (Lupus exulcerans fungosus), or they may tend rather to a rapid destruction of the tissues (Lupus exedens, vorax). The disease most frequently attacks the face, especially the nose, cheeks, and lips. The most frightful destruction is caused by it. The whole nose may be lost from lupous ulceration, also the lips. I have seen a case in which the whole skin of the face, the nose, the lips, and the eyelids, were destroyed by it; both the eyes had been lost from suppuration, and the exposed portion of the face belonging to the skull presented the most frightful appearance. Dieffenbach described such a case in a Polish Countess, and compared the appearance of her face to that of a skull. Lupous ulcers present no thoroughly constant phenomena, but their circumference and the general picture of the diseased portions of the skin, facilitate the diagnosis in a high degree. Only when lupus occurs on other parts of the body, *e. g.* on the extremities, or on mucous membranes, in the throat, on the conjunctiva, &c., does the diagnosis become difficult and not always certain. On the extremities, we may not only be excused but frequently can scarcely avoid confounding it with certain forms of lepra, and in the throat, with syphilitic ulcers. Lupus must, in many cases, be regarded as a constitutional affection which becomes

localised in the skin. Whether we should be justified in assuming the existence of a special lupous dycrasia is doubtful, since lupus very often develops itself in scrofulous individuals, so that we may set it down as a very malignant manifestation of scrofulosis. Lupus occurs further as one of the phenomena of syphilis, so that a Lupus syphiliticus and a Lupus scrofulosus have been recognised. Lupus most frequently occurs in the years of puberty, and is more common in females than in males. It is more rarely developed in later years; after forty there is but little danger of its occurrence.

As regards the *treatment*, I attach the greatest importance first to the local treatment, especially in the ulcerative form, since our object must be to check the spread of the mischief by all the remedies at our disposal, because the whole skin of the face is endangered and the internal remedies act very slowly. The question is here, as in all rapidly spreading processes of ulceration, to effect a radical destruction of the floor and edges of the ulcer, followed by cauterisation extending into the healthy tissues. Nitrate of silver or caustic potash in the form of a pencil, which is pressed into the portions of skin softened by lupus, is most frequently used. Caustic applications in the form of paste may be employed, especially chloride of zinc paste, which is most simply made by mixing the chloride with flour or starch and stirring it into a paste with a few drops of water. This is to be smeared on to the ulcer. To attain our object more speedily and to render the action of the caustic more intense, it is better to scrape off the floor of the ulcer with a small, sharp-edged spoon (Volkmann), and when the hæmorrhage has been checked, then to apply the caustic. Of the various caustics in use I prefer caustic potash, because it combines most quickly with the tissues, and the pain, therefore, is soonest over. It may be applied under chloroform, so that the patient, on awakening, experiences only a slight and bearable burning sensation. Nitrate of silver continues painful longest, but, since it melts more slowly than caustic potash, has decided advantages for the cauterisation of various parts of the body. If the caustic action has been sufficient, a good granulating surface presents itself on the coming away of the eschar and cicatrises in the usual manner. Fresh lupus does not readily form in this cicatrix, but the cauterisation cannot prevent the development of new nodules in the neighbourhood. For the exfoliative and hypertrophic forms of lupus, painting with tincture of iodine is the

best local remedy. It is better to render its action less intense by the addition of a little glycerine. I have seen nodules of lupus dry up under this treatment, but it does not prevent relapses. Lastly, the lupous portions of skin may, in many cases, be excised with advantage.

As regards internal remedies, the only one from which I have seen good results is cod-liver oil, to the extent of 4—6 tablespoonfuls daily, but such a treatment must be continued for years. Courses of treatment with decoctions of sarsaparilla, &c., are efficacious only in Lupus syphiliticus. Arsenic, so useful in other chronic skin affections, does scarcely any good here. Lupus occurred very rarely in Switzerland. My experience of it is based chiefly upon the Berlin clinique, and if I must give you my opinion concerning the action of internal remedies, it will be that, in many cases, the lupous dyscrasia, like the scrofulous, dies out of itself in the course of years, but is incurable in some cases.

3. *Scorbutic ulcers.*—In scurvy, extravasations of blood occur at various points of the skin, and more especially of the muscles; the gums swell and assume a bluish-red colour, while ulcers form upon them which bleed readily. Hæmorrhage from the intestines, gradual emaciation, and weakness supervene, and many of these patients die in a miserable condition. In this severe form scurvy occurs, especially endemically on the coasts of the German Ocean, and in sailors who make or have made a long sea voyage. In the latter case the disease is attributed generally to the continued use of salt meat. In the interior a kind of acute scurvy occurs to which belong the morbus maculosus, purpura, and the like. Scurvy localised in the gums and mucous membrane of the mouths of children is very common in all countries: the gums swell and assume a dark bluish-red colour, bleed on the slightest touch, and ulcers form on them, which are covered with a yellow, greasy layer consisting of pus, fungi, and shreds of tissue. This form of the disease may, when it occurs in this way and is treated early, generally be cured quickly. The gums must be painted twice a day with a mixture of hydrochloric acid, or borax, and honey, mineral acids be given internally, and a light digestive diet be ordered. If these remedies are employed conscientiously the disease will very soon disappear. Endemic scurvy is very difficult to cure, especially because it is impossible, in most cases, to withdraw the patients

from the deleterious endemic conditions. The treatment with acids is strongly recommended here also.

4. *Syphilitic ulcers.*—The signs which are generally mentioned as especially characteristic of syphilitic ulcers refer chiefly to the primary ulcer of chancre, to soft chancre. This begins as a bladder or pustule, and develops itself into an ulcer of about the size of a pea, with a reddened circumference and yellowish, lardaceous floor. The ulcer of indurated chancre has a different appearance. A nodule is first formed in the skin of the glans or prepuce, and this nodule ulcerates from the surface after the manner of other cutaneous ulcers. It generally assumes an atonic, indolent character, often with a preponderating tendency to decay of the tissues. Broad condylomata, one of the milder manifestations of constitutional syphilis, have a decidedly exuberant character, and present, strictly speaking, nothing else but small, superficial, very circumscribed, fungous ulcers of the skin, which occur especially on the perineum, about the anus, and on the tongue. The cutaneous ulcers which present themselves in the later periods of constitutional syphilis often have a highly indurated, brownish-red circumference, are circular or of the shape of a horse-shoe, and are again rather atonic in character. Their site (forehead, lips, anterior surface of leg) is also characteristic. They always first commence as nodular, flat infiltrations which decay from the centre. The treatment of syphilitic ulcers must be chiefly internal and directed against constitutional syphilis. Locally we must employ powerful caustics if there be a tendency to very rapid spreading.

Earlier surgeons distinguished a series of forms of ulceration not yet mentioned here which were believed to be characteristic of their causal moments. Thus you find that Rust, in his work upon ulcers, wrote of rheumatic, arthritic, hæmorrhoidal, menstrual, abdominal, herpetic, and other ulcers. But I, equally with other surgeons of more recent times, have been unable to penetrate into the mysteries of this kind of diagnosis. It is now pretty generally recognised that these views were based rather upon an artificial system which had its roots in the older, humoral pathology than upon critical observation. If we are quite free from prejudice in our observations, we must indeed admit that certain forms of ulceration, especially when they occur in certain localities, permit of an inference as to their causal moments, but the appearance and form of the ulcers again depend greatly upon the anatomical

conditions of the diseased parts, as well as upon the distribution of the fibres in the skin (Wertheim), and upon the most varied external influences, so that we should be exposed to many deceptions and errors if we regarded the appearance of an ulcer too certainly as an always correct expression of a specific, constitutional cause.

LECTURE XXXI.

CHAPTER XVI.

ON CHRONIC INFLAMMATION OF THE PERIOSTEUM AND THE BONES AND ON NECROSIS.

Chronic periostitis and caries superficialis. Symptoms. Formation of osteophytes. Osteoplastic, suppurative forms. Anatomical characters of caries. Ætiology. Diagnosis. Combination of different forms.

GENTLEMEN,—Chronic inflammations of the bones and periosteum, to which we now pass on, are much more frequent than the acute forms; most frequent of all is *chronic periostitis,* which is not unfrequently combined with *ostitis* (*caries*) *superficialis.* In the earlier stages it may become dispersed, or may run into suppuration, with the formation of ulcers on the surface of the bone. This is mostly accompanied by a deposit of newly formed bony matter on the surface of the bone; long-continued periostitis will never remain without influence upon the bone.

Let us first take into consideration the *symptoms of a chronic periostitis.* Slight tenderness and moderate swelling of the immediate neighbourhood of the affected bone will, in most cases, be the first symptoms, with some disturbance of function, especially if the disease occur in one of the extremities. Spontaneous pain is generally very slight, or may be wanting altogether; pressure causes more severe pain, and we shall find at the same time that the mark of the finger remains visible on the skin for some time, showing that the tumidity of the skin is chiefly of an œdematous character. Things may remain at this stage for a long time and then return to their former condition just as gradually. The part to be especially regarded as affected in such a case is the external, loose layer of the connective tissue of the periosteum; it is in this

that the dilatation of the vessels and the serous and cellular infiltration occur.

Perfectly similar symptoms to those just described may, however, exist in a case of periostitis which is at the same time combined with *ostitis*, except that, in the latter case, the spontaneous pain is sometimes more intense. Violent, stabbing, tearing pains may also occur in the night. When such a process has continued for months and then entered upon a retrograde course, the affected bone is found to be thickened and uneven on the surface. If you have an opportunity of examining such a case anatomically, you will find that the two layers of the periosteum cannot be clearly distinguished from each other, and that both have been converted into a lardaceous-looking mass of pretty firm consistence. On examination microscopically, you find that this consists of connective tissue containing numerous cells, and traversed by capillary vessels more or less increased in number. This diseased, thickened periosteum can be torn from the surface of the bone more easily than in the normal state. The bone beneath it (I take the case of a cylindrical bone, *e.g.* the tibia) is covered on its surface with little projections of a peculiar, sometimes stalactitic form. If you now saw through the bone at this point, you find that these projections upon the still distinctly recognisable surface of the compact cortical substance consist of a layer of porous, evidently newly formed bony matter of varying thickness according to circumstances, which is, indeed, very closely connected with the cortical substance itself, but which may, if the process is not of too long standing, be chipped off with a chisel in connected pieces. If the process be of long standing, and the union be already very close, we find that the porous bony mass deposited has become compact, especially if the process of disease be really completed.

Let us pause for a moment at this stage of the process, and ask whence this *newly formed bony mass* came. It may have been formed on the under surface of the periosteum either by this or by the surface of the bone. The former is the most general theory, and we see therein a freshly excited activity in the periosteum such as existed before the growth of the bone was completed, when fresh masses of bone in regular layers are continuously formed on the inner surface of the periosteum. We may term this form of periostitis, combined with the formation of *osteophytes*, *osteo-plastic*, a name which I shall employ for brevity's sake. I do not, however,

agree with the view just mentioned, that osteophytes proceed from the periosteum alone, but am convinced that they really grow from the surface of the bone, as the Greek name indicates. Microscopical examination shows, namely, that in this case also, as in that of suppuration and development of granulations on the surface of bone, the connective tissue surrounding the small in- and outgoing vessels is the seat of the new formation, which proceeds from the Haversian canals opening on to the surface of the bone, and forms the first point of deposit for the new formation of bone, which then extends under the periosteum. These ossifying granulations grow from within outwards to a certain extent into the periosteum, and the latter then first, as it appears to me, takes a secondary part in the whole process. The shape of the osteophytes, which is very peculiar, depends upon the shape of the network of vessels about which the new formation of bone was deposited.

I have no intention here of disputing the well-established fact that the periosteum also, as well as the other parts lying near the bone, are capable of producing new bony matter, but I wish to point out that the osteoplastic periostitis, more strictly speaking, is an osteoplastic ostitis superficialis. Practically, this more subtle distinction has, as yet, no particular value. *Osteophytes are the product of an inflammatory irritation of the periosteum and surface of the bone; they are exactly what we call callus in fractures, and are formed in a similar manner.* I will take this opportunity of remarking that periostitis running its course with formation of osteophytes without suppuration is peculiar to many forms of constitutional syphilis. The pains in the bones which are occasionally so extreme in the head and shins in inveterate syphilis depend almost always upon osteoplastic periostitis and ostitis.

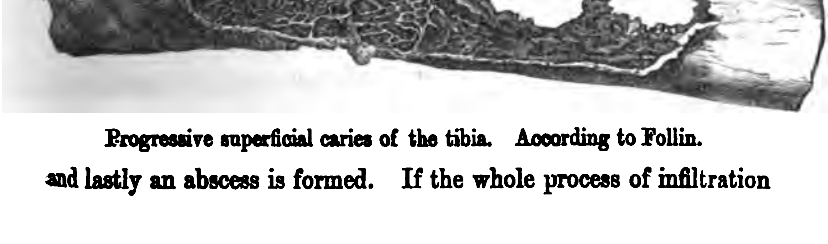
According to my observations, almost every chronic periostitis is, at first, osteoplastic; all other terminations commence sooner or later therefrom. In other words, *the chronic-inflammatory disturbance of nutrition in the periosteum and on the surface of the bone does not lead directly to the destruction of the tissue, but first causes a cellular infiltration, which is followed immediately by the formation of the tissue.*

Next in frequency is the *suppurative form of periostitis*. This may run its course without essential implication of the bone. Recall to mind the symptoms mentioned formerly—œdematous swelling of the skin, pain on deep pressure, to a slight extent also with

movements of the limb. This state of things continued for a long time unchanged, then the swelling gradually increased, and a tumour of the consistence of dough was formed, immovable and tolerably well, but not altogether defined. The skin also gradually became red, and the tumour presented a distinct feeling of fluctuation. This may have occupied perhaps from four to six months, and the tumour may again remain for a long time unchanged. The pain will probably have become somewhat greater, and the function of the limb be more interfered with. If we leave things entirely to themselves, the indolent abscess which has evidently been formed will burst, and thin pus mixed with flakes come away. If you introduce a probe into the narrow opening of the abscess, you arrive at a cavity lined with granulations. If you do not wait for the spontaneous opening of the abscess, but previously make an incision into the thin skin, it is possible that no pus may come away, but that you will find the very distinctly fluctuating tumour to consist of a mass of gelatinous, red granulation. In other cases, again, the whole tumour consists of pus.

You will easily understand, from what I told you formerly of the anatomical conditions in chronic inflammation, these differences met with on opening inflammation-nests. Represent to yourselves the occurrence, in a serous and plastic infiltration of the periosteum, of a copious development of vessels accompanied by infiltration of migrating cells and conversion of the connective tissue into a gelatinous intercellular substance. This becomes converted into a mucous mass of granulations, and sooner or later, perhaps, into pus,

FIG. 83.



Progressive superficial caries of the tibia. According to Follin.

and lastly an abscess is formed. If the whole process of infiltration

affects the periosteum and adjacent soft parts only, the bone remains tolerably unchanged; some tendency to new formation of tissue shows itself in the production of a layer of osteophytes under and in the periphery of the periostitic nest. The possibility exists, however, that the abscess may slowly heal after the discharge of the pus, and the previous normal state of things be almost re-established. Such a periostitis leading to the formation of an abscess without implicating the bone sometimes occurs in practice, but this is rare. It much more frequently happens that the bone also becomes diseased, although only superficially; that, therefore, ostitis accompanies the periostitis, and not an ossifying, but a chronic, suppurative, ulcerative ostitis, a *caries superficialis*. The symptoms of such a caries are, previously to the bursting of the abscess externally, scarcely different from those of suppurative periostitis. If the abscess be opened, however, we can apply the probe to the surface of the bone and feel the eroded, rough, friable bone. But the caries existed long before the abscess was opened, the process penetrated stealthily into the bone itself; it existed perhaps already when the periosteum appeared to be infiltrated only, when it was still in the stage of gelatinous granulations. Suppuration is, therefore, not necessarily combined with caries, although it often supervenes thereupon. To render all this clearer to ourselves, we must study chronic ostitis in preparations: the whole mode of development and aftercourse is completely analogous to the chronic inflammatory process in the soft parts, but the hardness and difficulty of solubility of the bones furnish somewhat different conditions.

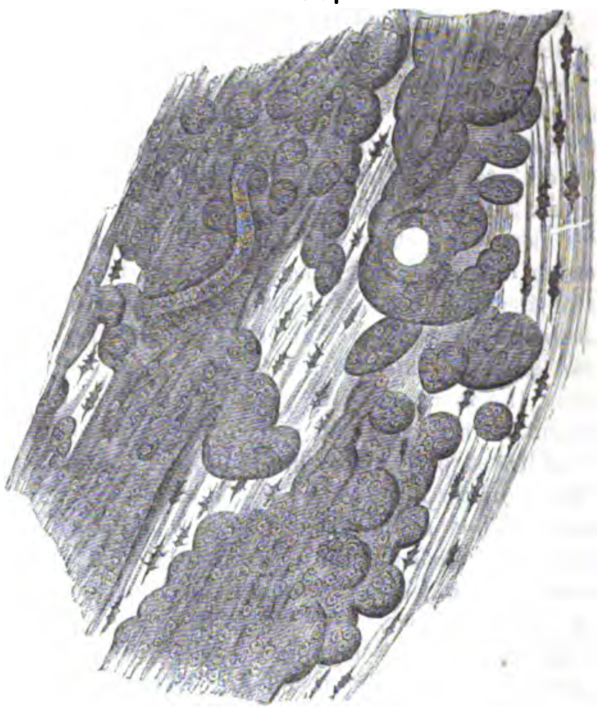
We have in the course of these lectures very often repeated already that the new formation of tissue occasioned by the inflammatory disturbance of nutrition develops itself in and from the affected tissues, that the tense fibres of connective tissue become converted by copious cellular infiltration into gelatinous, or even into fluid intercellular substance. How can this be accomplished in bone? The star-shaped bone corpuscles, so long as they are firmly enclosed in the calcareous mass, present as little of proliferous exuberance as do the stable corpuscles of connective tissue in the field of inflammation. The inflammatory newgrowth infiltrates itself here, as in most of the tissues of the body, into the connective tissue, and in fact into the connective tissue which surrounds the vessels of the bone lying in the Haversian canals and medulla. But

the space for the copiously formed cells is narrow, and if the cell-migration should occur very energetically, the vessels in the bony canal would become thoroughly compressed thereby. If the circulation then ceased, the nutrition of the young cells would also therewith cease, and death of the corresponding portion of bone (necrosis) necessarily result. Or the disturbance of nutrition in the bony tissue might also be so great from other causes that death of the part would ensue. It is quite true that this may happen; superficial necrosis may be combined in this manner with periostitis, of which more later on. Generally the cellular infiltration in the Haversian canals is not so rapid as to compress the vessels; we have to deal here with a chronic process; the bone gradually yields, the Haversian canals become wider and wider, the firm cortical substance of the bone becomes porous, and in the meshes of the dilated canals lies the young crop of cells, provided with gelatinous intercellular tissue—*an interstitial, intra-ostal exuberant process of granulation*. If you represent to yourselves the process as going on further and further, more and more bone disappears, and even the whole infiltrated portion may break up, and the place of the bone be taken by the inflammatory newgrowth. If you macerate such a bone, you find at the diseased point a defect with rough, porous, as it were eroded walls. In this defect lay the inflammatory newgrowth which had consumed the bone and taken its place (Fig. 55). Observe here that there is, as yet, no question of pus, but the inflammatory newgrowth may of course suppurate later on, and if we remain true to our assumption that the process commenced in the periosteum, we have an *indolent abscess* lying superficially upon the bone, the walls of which may be completely lined with granulations.

If you have followed me carefully so far, you will already have remarked that the bony tissue remains altogether passive in the whole process; it becomes consumed, and we might to a certain extent be justified in asserting that the chronic ostitis or caries is, strictly speaking, only a chronic inflammation of the connective tissue in the bone, with solution of the latter. The inflammatory disturbance of nutrition in the bone declares itself only thereby that the salts of lime gradually disappear, and its organic framework then becomes absorbed by the vessels of the inflammatory new growth. I do not therewith deny the disturbance of nutrition in the bone, nor yet the ostitis, but only wish to point out that in the

histopoietic processes of ostitis the bony tissue itself takes no part. This is my view, but there are many surgeons and anatomists who do not agree with it. None of the works hitherto published on

Fig. 84.



Transverse section of a piece of carious bone. Caries granulosa seu fungosa Magnified 350 times.

the subject have convinced me that I am wrong. Now, how is the destruction of the bone effected? Should not an examination with the microscope give us some evidence whether the bone cells undergo a change in the process or not? If you take a minute portion of bone, the thinnest possible plate from a carious nest, with the forceps and examine it under the microscope, you will find the edges and surfaces in many places eroded as it were. The bone corpuscles are unchanged; the intercellular substance is, perhaps, somewhat more turbid than usual, but does not show any marked change; a slice of bone from the neighbourhood of such a carious nest presents nothing more than this. If you saw or cut out a portion of a carious nest, and deprive the bone gradually of its

salts of lime by means of chromic acid, and then make incisions through it, which you clear with glycerine, you obtain something closely resembling the accompanying representation (Fig. 84).

The pieces of bone often appear eroded at their edges in a pretty regular manner; into these defects grows the new formation, the further development of which goes on concomitantly with the solution of the salts of lime and the absorption of the organic framework of the bone; the bone corpuscles remain unchanged. What becomes of the cells contained in them is doubtful; they are not distinguishable from the innumerable young cells of the inflammatory new growth with which they mix. It is possible that, once freed from confinement, they contribute, by division, to the crop of cells, as some observers (O. Weber, Volkmann, Heitzmann, and others) have assumed, or they may perish altogether. In any case, so far as we can judge from the changes in form generally, they do not contribute to the solution of the bone. By what means the bone becomes dissolved here is a problem hitherto unsolved. Living as well as dead bones may, to a certain extent, be consumed by the interstitial bony granulations. I told you formerly, as you may remember in connection with the operation for pseudarthrosis by means of ivory wedges driven in, that these wedges become rough and carious on their surfaces. The process is exactly the same there, and this observation is especially interesting and important as supporting the view that the bone to be absorbed does not necessarily contribute to its own process of solution in caries, but may play an entirely passive part therein. Although the granulations adhere closely and firmly to these gaps in the ivory wedges, they certainly do not spring from the dead ivory, but from the irritated bone around it. We very frequently find in these granulations, especially in the vicinity of a lacunar erosion, giant cells with numerous nuclei very freely developed. Kölliker, who found these cells also in the medullary canal of growing bones, where, as I have pointed out already, lacunar erosions occur as the expression of a process of absorption, places them in very close connection with the absorption of bone, and calls them, on that account, *osteoclasts*. Wegener demonstrated that these giant cells develop themselves especially from the walls of the vessels, and produce the small holes which are so often observed on the inner surface of the skull through absorption of the bony substance by the action of the vessels of the Pacchionian

granulations. To meet the objection which might be made that I admit this form *alone* of absorption of bone, in which the formations described occur on the surface, I must here remind you that I formerly called attention to the fact that the ivory wedges used in the operation for pseudarthrosis do not *always* become rough on their surfaces, but may remain smooth and yet have lost in weight, as may be shown by weighing them before and after the operation.

The above description of the morphological changes in carious bones, which R. Volkmann very graphically calls *lacunar corrosion*, and which were first made known by Howship, is now very generally recognised as correct, although other views are also entertained on the subject which, if specially interested therein, you may find in the cellular pathology of Virchow, in Förster's 'Atlas,' and in the classical works of Volkmann on diseases of the bones.

There is one thing, however, which we must not overlook: it is quite possible, namely, that the bony substance, having its nutrition interfered with, might begin to break down in minute particles in the form of very fine powder; this might happen very easily if the bone were previously deprived of its organic substance. It might be argued, in fact, that this is the primary process in *ulceration or caries of bone*, and those who, in the case of ulcers of the soft parts, regard the decay of the tissues as the primary, the inflammatory new growth as the secondary process, will take the same view also in the case of the bones. My observations are distinctly opposed to the generalisation of such a theory of the process of ulceration, as I have already told you, and I cannot accept, for the case of the bones, what I have not found reliable for the soft parts. There is not the least doubt, however, that individual portions of bone break up and that, in a case of suppurative ostitis, such small particles of bone are found in the pus. We should here have to do with a necrosis in the smallest form; such a death of individual particles of tissue occurs, in fact, in the soft parts, as well in the acute as in the chronic process of inflammation. You will no doubt remember that we have already spoken of this; as a rule, it cannot be shown in caries, but occurs occasionally in caries with suppuration or caseous degeneration. In such cases it may happen that even larger pieces of bone become actually necrotic, and this combination of caries with necrosis has received the special name of *caries necrotica*.

We have hitherto employed the term *caries* as altogether synonymous with chronic ostitis and breaking up of bone, and such is

now the very general custom. Formerly, however, the term caries was used only for the process of ulceration combined with suppuration, for *open ulcers of bone.* The intimate connection between chronic inflammation and ulceration, which we showed at an earlier period in the soft parts, exists also between chronic ostitis and caries. It would be better, perhaps, to abandon gradually the term caries altogether and to replace it by ostitis with various predicates, such as rarefying, osteoplastic, ulcerative, granular, &c.; or to employ the term caries only for losses of bone caused by lacunar erosions. In macerated bones this is always easy to recognise; we are there never in doubt whether we shall call a given bone carious or not, for we call all those losses of substance carious which appear eroded. We might call them very properly *lacunar* or *corrosion-defects.* On examination during life, however, it requires exact knowledge and great experience to distinguish with certainty whether a bone into which we can easily pass a probe is merely softened or whether it presents larger lacunar defects.

We have as yet become acquainted with superficial caries only, later on we shall come to central caries also, which stands in the same relation to the superficial as a cavernous ulcer to an ulcer on the surface. You have first become acquainted in bone with an *ostitis fungosa* or *granulosa* (Virchow's and Volkmann's *caries sicca* signifies caries with exuberant granulations and destruction of bone, without suppuration), in which there was not yet any question of decay of the chronic inflammatory new growth, but in which the bone is traversed by interstitial granulation tissue (granuloma). *This is by no means always the case to such an extent* as we have now assumed. If you bear in mind the atonic, indolent ulcer in the soft parts, how the new growth there rapidly becomes purulent or caseous, or undergoes molecular decay, and apply this simply to the new growth in the bone, you will easily understand what goes on. The caries then assumes a different character; there are very indolent, atonic forms of caries, in which the new growth causes the solution of only a very small quantity of the bony substance, and then decays or becomes caseous, and thus *a kind of maceration of the diseased bone occurs in the living organism.* The soft parts in the bone suppurate. If this occur *before* the bone is dissolved, the piece of bone suppurated out becomes *necrotic.* Deficient vascularity in the new growth plays here also the chief part in the decay. But why, in one case, a fungous, exuberant, in another an atonic

caries results is a circumstance the causes of which must be looked for in the diseased organism itself.

We shall soon become acquainted with still other forms of ostitis when we have to speak of chronic inflammations occurring primarily in the bone itself.

Chronic inflammation of the periosteum and bones has its causes chiefly in constitutional affections, and if an injury (a fall, blow, &c.) may become the incidental cause of such diseases, the chief element must still lie either in the part injured or in the whole organism, for otherwise the process would run its usual course, as in all traumatic inflammations, and soon come to a termination. If a traumatic injury causes lingering, chronic processes of inflammation, this must result either from an altogether peculiar local disturbance difficult to remedy, or from a general tendency. I see no reason, as yet, for abandoning these views propounded at an earlier stage. It is syphilis and scrofulosis especially which dispose to chronic periostitis and ostitis, and in general the fungous forms of caries are more frequent in children, the atonic in adults. True tubercles also occur in bone, but not, so far as I know, in the periosteum or in the cortical layer of the cylindrical bones. But chronic periostitis also occurs very frequently where no such dyscrasia is demonstrable, nor, indeed, any other special cause; in old people especially, periostitis with caries often occurs after very slight injuries, and in the most disagreeable indolent forms. The inflammatory new growth in the bone will participate essentially if the whole organism collapses; in children who have died of caries you will almost always find the atonic forms, for, a short time before death in them, when nutrition was already impaired, the new growth also collapses, and the diseased bone becomes macerated during life from suppuration. Pathological anatomists, who see caries in the dissecting room only, seldom know the granulo-fungous form well, or regard it as the less frequent one. But any one who has frequent opportunities of examining pieces of carious bone removed during life, especially of resected joints in children, where the process is still in a state of such active development, will form a very different opinion from that based upon anatomical collections, in which macerated bones only are generally preserved. If I have here spoken of fungous and atonic caries only, you are quite aware that I mean to designate thereby only the extremes of the exuberant and the rapidly decaying forms of new growth;

between the two lie, of course, various degrees of vitality. It is not the object of these lectures to describe all the existing shades of this process, as will be done in the clinique, but to render clear to you, in distinct types, the forms of disease; you must first of all acquire a clear comprehension of the material in its most general sense, and on that account I only make you so far acquainted with the details of these processes as appears to me necessary for a correct conception of them.

Now, whereby can we recognise whether a given carious process, which we have as yet diagnosed by means of the probe alone, has more of an exuberant or of an indolent character? This is a question you may fairly ask, since it cannot fail to influence our treatment, as in the case of ulcers of the soft parts. It is important not only for the treatment but also for the prognosis, for thoroughly indolent caries offers much worse chances than the fungous form, if only because it most commonly occurs in wretched, ill-fed subjects and in old people. The distinction is not difficult: in the more exuberant forms the swelling of the soft parts, the periosteum, the skin, and especially of the capsules of the joints when the caries is in them, is often very considerable; all these parts feel of a spongy softness. If there are openings in the skin, exuberant granulations project from them and there is a discharge of mucous tough pus resembling synovia. If you make an examination with the probe, you do not at once feel exposed bone, but must push the probe into the granulations, and often pretty deeply, to penetrate into the friable bone. In the truly atonic forms the skin is thin, red, and often undermined. The edges of the openings are sharp, as if cut out with a punch, and discharge a thin, serous, sometimes ill-smelling, or ichorous pus. The skin and cellular tissue are often highly œdematous, and if you introduce a probe you come at once on to the exposed, rough bone, the soft parts of which have already been macerated out by suppuration. This is the state of things in the most extreme cases of a long series; many intermediate forms lie between.

If you take all these things into consideration you will now, I think, have formed a correct notion of periostitis and caries superficialis.

Let us sum up briefly what we know at present of the chronic diseases of the periosteum and bones. We have had under consideration chronic osteoplastic periostitis (with formation of osteo-

phytes but no suppuration); further, simple suppurative periostitis; further, the latter combined with caries superficialis. But osteoplastic periostitis may also be combined with suppurative periostitis and ostitis, and this combination is, in fact, not uncommon, *i. e.* osteophytes are formed around a carious nest in the bone. If we examine a series of preparations of carious joints, we find, round about the destroyed parts, osteophytes springing from the surface of the bone; the periostitis, which led at one point to destruction of the bone, caused the new formation of bone in the vicinity. You may very fairly compare this with an ulcer with callous edges: thickening from new growth in the periphery, decay in the centre. It is not in the atonic forms of caries, however, that we meet with considerable formations of osteophytes in the periphery, but only in those which, for some time at least, bore the exuberant character, just as, in indolent, scrofulous ulcers of the skin, no thickened edges occur, but only where the skin had been for a considerable time previously plastically infiltrated and thickened. In the bones, therefore, we meet again with this combination of exuberance and decay which we have already so often observed in inflammation.

LECTURE XXXII.

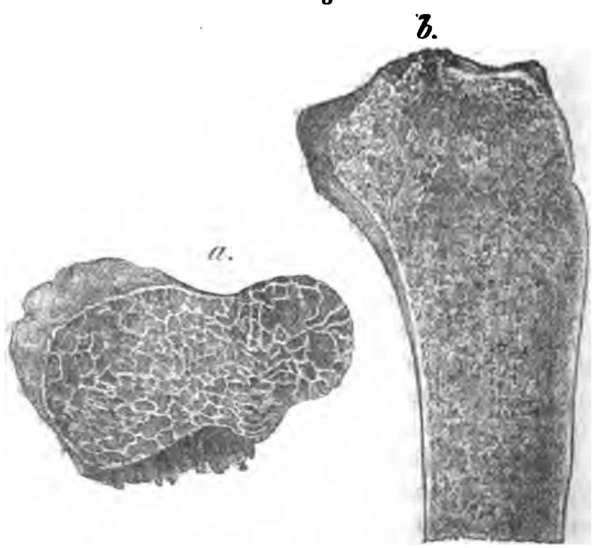
Primary chronic ostitis: Symptoms. Ostitis malacissans, osteoplastica, suppurativa, fungosa. Chronic osteomyelitis. Caries centralis.—Abscess in bone. Combinations. Ostitis with caseous degeneration. Tubercles in bone.—Diagnosis. Displacements of the bones after partial destruction of them.—Abscesses from congestion.—Ætiology.

We have hitherto spoken of chronic ostitis only in so far as it is dependent upon periostitis; this will be the case in the cylindrical bones, since the cortical layer in them has but little tendency to become diseased primarily, except perhaps in syphilis. It is otherwise, however, with the spongy bones and spongy parts of bones. A chronic inflammatory process may be set up in them just as, in the medullary canal of a cylindrical bone, a circumscribed chronic osteomyelitis may occur and implicate the cortical substance from within. These cases are called simply *ostitis;* this may lead to *abscess in the bone,* and finally to *caries centralis.* The symptoms of such a deep-seated, chronic inflammation arising in the bone are, in many cases, very slightly characteristic at first. A dull, moderate pain, causing a slight disturbance of function, is often the only symptom. Swelling does not come on until late, and the disease may have existed for months before we are in a position to form a certain diagnosis. But if more severe pain on pressure and œdema of the skin supervene, and the periosteum becomes involved secondarily in the chronic inflammatory process, we are gradually led to a correct diagnosis, and that the more easily if the process be circumscribed and finally opens outwards so that we can penetrate with the probe deeply into the bone, and thus directly recognise the disease. In many cases the periostitis is, for a long time, the chief symptom of the ostitis; the former may become so prominent as to appear to be the only disease, until, first from the long duration of the process, then from defects which arise in the bone from within outwards and, perhaps, finally from the coming away of small pieces of bone, our attention is attracted to the fact

that the long-continued suppuration has its cause in a more deeply-seated affection of the bone.

I have already informed you that the chronic inflammatory disturbance of nutrition in the bone first declares itself by a chemical decomposition of the latter, by which its salts of lime are rendered soluble. We have hitherto had under consideration cases only in which the disease was confined to circumscribed spots, and penetrated from without inwards. Imagine to yourselves now an ostitis becoming developed in a spongy bone (in one of the tarsal bones, or in the diaphysis of a cylindrical bone, *e. g.* in the inferior diaphysis of the tibia) in which the salts of lime disappear from the bony tissue, while the vessels of the medulla become more numerous, and the medulla, infiltrated with migratory cells, takes the place of the gradually but constantly decreasing bony tissue. We have here the picture of a genuine *ostitis malacissans*, an osteomalacia inflammatoria, or, according to Volkmann, a rarefying ostitis. The

FIG. 85.



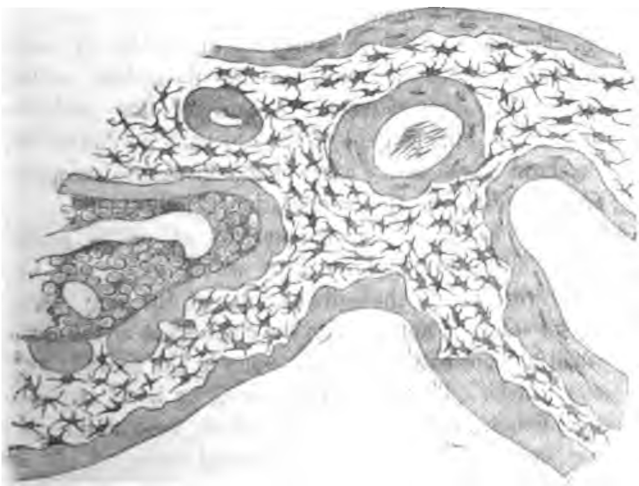
Ostitis malacissans. *a.* Vertical section of calcaneus diseased at its anterior and posterior end, normal in the middle. *b.* Vertical section of upper end of tibia, rather highly porous.

bones then become extremely light, and their cortical substance very thin.

Rindfleisch has shown how the wasting takes place under these

circumstances by demonstrating that the salts of lime first become dissolved and disappear in the same manner as in lacunar corrosion. But while, in the latter, the bony tissue disappears at the same time as the salts of lime of the bone, in the case before us the bony tissue remains for some time in its skeleton form. It is evident from old cases, in which every trace of bony tissue within the periosteum has thus finally disappeared, that the bony tissue itself, after it has been deprived of its salts of lime, at last also becomes absorbed. But whether this is always the case, or whether it can again become impregnated with salts of lime and again become converted into normal bone, must at present be left an open question; as yet we know nothing on the subject.

Fig. 86.



Wasting of the salts of lime out of the peripheric portions of the bony structure in ostitis malacissans. Magnified 350 times. According to Rindfleisch.

Whether this kind of wasting, which may be correctly termed *halisteresis ossium* (from ἅλς, salt, and στέρησις, deprivation, Kilian), always occurs in the manner shown in fig. 86, has not yet been sufficiently investigated; it might happen that in this wasting of the inflamed bony tissue the salts of lime and the tissue were absorbed simultaneously. That in the bone corpuscles of the tissue deprived of lime no trace of exuberant growth is discoverable appears to me a further proof that the bone cells have no tendency to proliferation.

We have here, therefore, a form of inflammation of bone in which

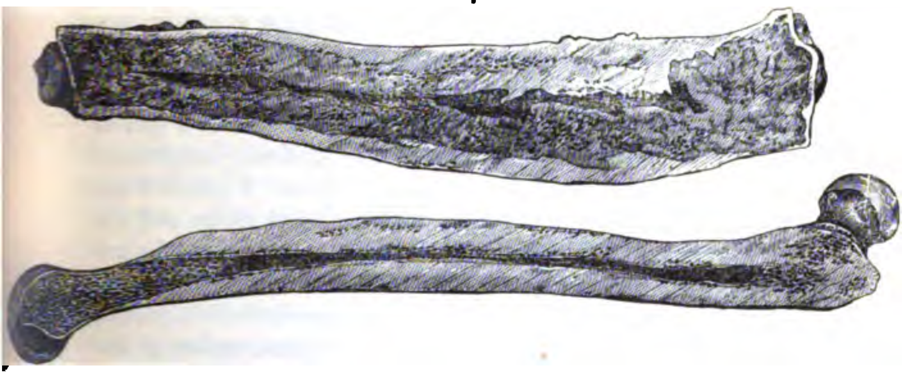
the wasting thereof is a prominent feature. In the bone also there is generally only a very slight formation of osteophytes, and even this may be altogether absent. In the interior of the bone no regenerative processes are set up; the medulla rendered highly red by increased vascularity remains, for the most part, fatty, but is more copious than usual in the bones of adults; it is also occupied by young cells, and thus resembles rather the medulla in the bones of children. In this state ostitis malacissans may remain for an indefinite time; by its gradual advance it would finally lead to the complete solution of the bone, so that only medulla and periosteum would remain, and the bone, on account of its softness,' yield to every force acting upon it; this is rare. According to my experience, it is equally rare for the medulla in these bones to undergo suppuration or caseous degeneration without some external cause; no doubt there are many such causes by which this is occasionally brought about—violent probing, the use of unclean probes, compression, operative interference. A cure of this ostitis by new formation of bone in the gaps of the old bone no doubt frequently occurs in slight cases; severe cases of this disease in atrophic individuals are incurable, and then amputation is indicated.

Osteoplastic ostitis is the counterpart of ostitis malacissans; whether the disturbance of nutrition which gives rise to it also commences with loss of the salts of lime of the bony tissue in the very earliest stages is not known; the chief effect of the disturbance is abnormal new formation of bony tissue in the medulla and in the Haversian canals. The disease, if it develops itself strongly in the cylindrical bones, generally affects the whole bone at the same time, and often occurs simultaneously in several bones of the skeleton. The consequence of such an affection may be the complete filling up of the medullary canal with a pretty compact bony mass, further the almost complete filling up of the Haversian canals with bony substance; formation of bone on the surface is also generally combined therewith.

The whole bone thus becomes enormously heavy and abnormally thick; this is commonly called *diffuse hypertrophy of the bone*, but still more frequently *sclerosis ossium* (R. Volkmann). Moreover not only cylindrical but other bones of the skeleton are occasionally affected, *e. g.* the bones of the face and of the pelvis. The bony deposits are then mostly spongy, puffy, and nodulated, so that such a bone resembles the skin when degenerated from

elephantiasis. These processes are, in fact, closely related to each other (*leontiasis ossium*, Virchow). The filling up of the diploë between the internal and external table of the bones of the cranium

Fig. 87.



Sclerosed tibia and femur: the former according to Follin; the latter a preparation from the pathologico-anatomical collection in Vienna.

with a bony mass is indeed such a very frequent change in these bones at an advanced age that it can scarcely be termed pathological, but belongs here, however. The causes of sclerosis as a primary process of disease are altogether obscure. Syphilis may, in many cases, be a causal element therein, but the bony formations which occur in that disease seldom acquire such a density as in true sclerosis. In rare cases only shall we be able to diagnose the disease with certainty during life, because these bones present to the feel nothing else whatever than a somewhat increased thickness and generally only a very slight unevenness of the surface.

Caries interna suppurativa circumscripta, i.e. the occurrence of lacunar defects in the interior of the bone, generally occurs primarily in a cylindrical bone as osteomyelitis. The inflammation-nest spreads gradually to the inner surface of the cortical substance; this becomes broken up, as we have already described, and finally, at one point, completely destroyed. In the centre of the inflammatory new growth pus may, in such cases, be formed rather early and eventually be discharged outwards. This is the disease which is specially designated as *abscess in bone*. The periosteum does not remain inactive therein, it becomes thickened, and on the surface of the bone irritated from within, but not yet penetrated, there is very

often formed, in this case also, a new bony deposit. The cylindrical bone thereby becomes thickened externally at the point where the abscess was formed in its interior, and thus it comes to appear as if the bone were burst open to a certain extent. It is difficult, often impossible, to distinguish such an abscess in bone during life from a circumscribed, osteoplastic periostitis, and we must, therefore, not be too ready with operative interference. This chronic, suppurative, central osteomyelitis may gradually extend to the whole medullary canal of the bone. I have recently seen such a case in a girl of fifteen: the medulla of the whole radius had gradually suppurated away and the cortical layer had become very thin. I resected the whole diaphysis, leaving behind the thickened periosteum and the two epiphyses; the wound healed in three months, but the regeneration was extremely slight.

With this central, carious process may be combined also a partial necrosis of individual particles of bone on the inner surface of the cortical substance, so that we have to do with a *caries necrotica centralis*. Lastly, there are cases of the very worst kind in which chronic internal and external caries are combined with necrosis and with partly suppurative, partly osteoplastic periostitis, partly condensing, partly rarefying ostitis, all in one and the same cylindrical bone. At various points of the bone, in such cases, abscesses are formed; we sometimes arrive with the probe at the friable bone, sometimes at a sequestrum; at one point we penetrate into the medullary canal, at another only the surface of the bone appears to be diseased. The whole bone is thickened as well as the periosteum, and the fistulous openings discharge thin pus. A macerated preparation of such a bone presents a very peculiar appearance; the surface is sometimes more, sometimes less covered with porous osteophytes; amongst these are found here and there necrotic pieces which belong to the surface of the bone; some openings lead into the medullary canal. If you make a longitudinal section of such a bone, you find the medullary canal also partly filled with a porous bony mass; the cortical layer has lost its uniform density and is also porous, so that it can be distinguished from the deposits of osteophytes at a few points only. In the original bony canal we here and there meet with rather large, roundish holes, in some of which are necrotic pieces of bone. These bones are in such a condition that a cure can seldom be expected in such cases, and either extirpation of the bone or amputation of the limb becomes necessary.

Very similar is the state of things in caries of the *short spongy bones;* in them an exuberant inflammatory new growth leads comparatively quickly to breaking up of the bone, generally with consecutive, often suppurative periostitis, although such is not always the necessary result. There are cases of ostitis of the short spongy bones of the hand or foot, and especially of the epiphyses of the cylindrical bones, in which, without any considerable amount of swelling (which generally first results from the supervening periostitis) the bones become completely broken up by the penetration of a mass of interstitial granulations, without the least trace of suppuration having been observed (*ostitis interna granulosa seu fungosa*). The consequence of such breaking of the bone in the joints just mentioned, as well as in others, is that the bones will be displaced by muscular action in the direction of the most complete destruction of the bone. From the distortions so caused we may judge approximatively also of the extent of destruction of the bone. Thus I was once obliged to amputate a foot which was so much distorted in consequence of such a destruction of bone without suppuration on the inner side of the talus and calcaneus that the inner edge of the foot was drawn upwards as in an extreme case of congenital clubfoot, and the patient walked with great difficulty on the outer edge of the foot. Moreover, a rather large ulcer had formed on the outer edge of the foot which finally rendered walking quite impossible. I have seen a similar case in the hand: a young woman of twenty had already suffered for a long time from pain in the left wrist without swelling of the soft parts; pressure upon the carpal bones caused severe pain. The hand gradually became strongly abducted without the occurrence of swelling or suppuration; the administration of chloroform rendered it possible to restore the hand to the normal position, when it became evident that a part of the carpal bones had entirely disappeared.

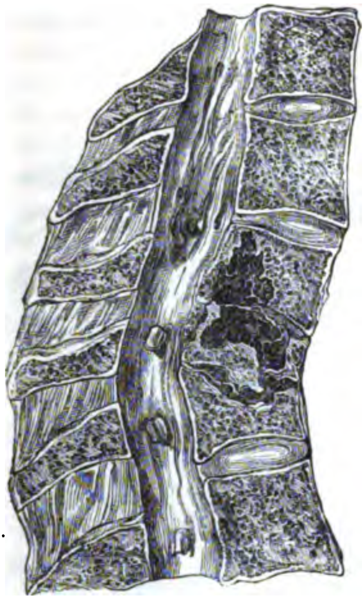
In the larger spongy bones, *e.g.* in the calcaneus and in the epiphyses of large cylindrical bones, a large central cavity, an abscess in bone may be formed, with which a central necrosis may be combined. In the very great majority of cases, however, the ostitis is combined with a suppurative periostitis, and this is most frequent in the small carpal and tarsal bones; these are so small that, if the periosteum become affected, the disease very easily spreads to the whole bone and its articular surfaces, and that, conversely, primary disease of these bones very rapidly reacts upon the

periosteum and the articular surfaces. Further, the sheaths of the tendons and the skin become involved, and the latter pierced at various points by ulceration from within outwards. In the case of the hand, the radius and ulna may then become involved, as well as the articular extremities of the metacarpal bones; in the foot, the lower end of the tibia and fibula and the posterior ends of the metatarsal bones. The joint of the hand or foot then becomes much swelled and deformed; at many points thin pus flows from the fistular openings, and the carpal or tarsal bones are partly destroyed and replaced by exuberant, spongy granulations, or are entirely or partially necrosed. I need scarcely call your special attention to the circumstance that the course of this form also of primary suppurative ostitis with caries is just as variable as regards its vitality as chronic periostitis, and that you can here also distinguish some cases which have a distinctly atonic, others a fungous character, while a series of cases lies between these extremes.

There is still one form of chronic ostitis to which I must specially allude, viz. *ostitis with caseous degeneration* of the inflammatory new growth, generally accompanied by slowly developed lacunar defects, and often by partial necroses. This kind of chronic inflammation is already known to you; it belongs generally to the atonic forms, with deficiency or total absence of vascularity. It occurs chiefly in the spongy bones; in the cheesy mass which fills the cavity in the bone are found pieces of bone almost always dead but not dissolved. The vertebræ, the epiphyses of the larger cylindrical bones, and the calcaneus are the most frequent seat of this *ostitis interna caseosa.* This form is recognisable in a few cases only; we gradually arrive at the diagnosis of internal ostitis, but can distinguish the special form in those cases only in which penetration outwards occurs, and the semi-fluid caseous matter is discharged. Ponfick has shown that circumscribed central ostitis not unfrequently becomes developed in typhoid fever. After exanthems also, such as scarlet fever, smallpox, measles, diseases of the bones not unfrequently occur which are in connection with the general acute process of disease. These forms of inflammation tend especially to caseous degeneration and caries necrotica. I must not omit to mention in conclusion that, in rare cases, generally in the vicinity of caseous nests, true *miliary tubercles* also occur, small, at first gray nodules, which afterwards undergo caseous degeneration, in the spongy substance of the epiphyses, in the

tarsal bones, and in the bodies of the vertebræ. A diagnosis of this true tuberculosis in bone cannot be made during life, and may,

FIG. 88.



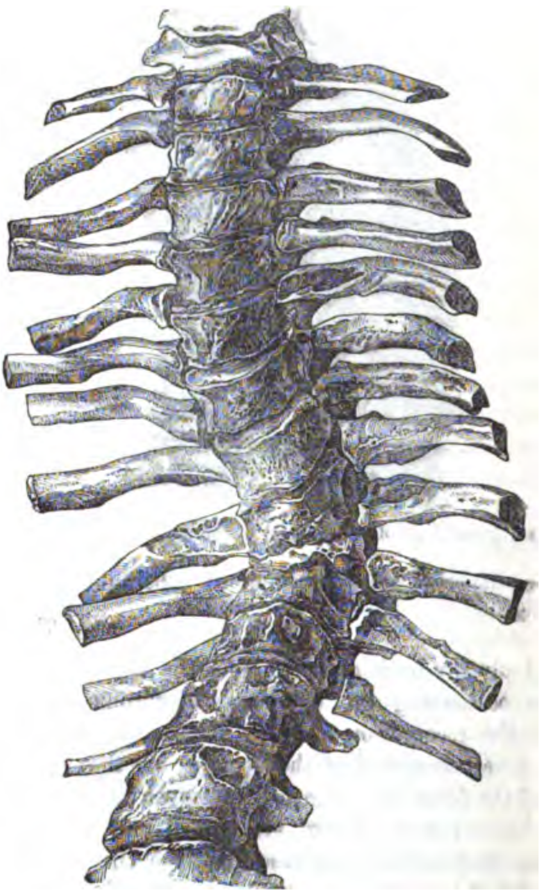
Caseous degeneration of ostitic nest in the dorsal vertebræ of a man.

at most, be suspected when pronounced tuberculosis of the lungs or larynx exists.

You will already have gathered from the few remarks which I have made concerning the diagnosis of chronic periostitis and ostitis that the recognition of these diseases generally is not very difficult at a certain period of their course, but that an exact determination of the form and extension in a given case does not always lie within our power. There are two points which essentially facilitate the diagnosis in those cases in which direct examination of the bone with the probe is impossible, viz. the *displacements of the bones* which must occur, at many points of the body at least, in consequence of their partial destruction, and the *formation of abscesses* which so often accompanies them. Carious destruction of the larger cylindrical bones will seldom become so extensive as to occasion a solution of continuity; where this might no doubt

happen it is often prevented by the circumstance that osteophytes are formed externally on the bone simultaneously with the process of destruction in its interior, and the bone thus becomes thickened at the point of disease. I have as yet seen only one case of thoroughly atonic caries of the tibia of an old and decrepid in-

FIG. 89.



Destruction of vertebræ by multiple periostitis and ostitis of their anterior surfaces. Preparation from the pathologico-anatomical collection at Basle.

dividual in which the bone was completely eaten through at one point, so that continuity was quite interrupted and a spontaneous fracture produced. In caries of the ribs I have twice seen

spontaneous fracture produced; the post-mortem examination showed no trace of formation of osteophytes. The bone represented by fig. 83 also is almost eaten through from without inwards. In the small cylindrical bones of the phalanges and metacarpus, a complete destruction of the bone not unfrequently occurs; scrofulous caries of these bones has long been called *pædarthrocacis* (from Greek words signifying an ill-condition of the joints in children), or spina ventosa, old names meaning nothing else than caries of the fingers or toes with spindle-shaped enlargement. If the bones become completely destroyed, partly from exuberant granulation, partly from partial necrosis of the small diaphyses, the fingers shrink up and are then drawn forcibly backwards by the tendons, so as to form unsightly rudimental fingers. Much more frequent is the displacement of the bones in the case of the spongy bones, when these are destroyed. I have mentioned this already when speaking of the carpal and tarsal bones, but it occurs in a much more extensive manner in other bones; if, for instance, the head of the upper edge of the acetabulum be destroyed by caries, the femur gradually becomes drawn upwards in proportion to the amount of destruction, and assumes a position similiar to that in a case of dislocation of the hip-joint upwards. Similar dislocations, although less striking, occur also in the shoulder-, elbow-, and knee-joints. Almost the most striking are the dislocations of the spinal column, after carious destruction of the bodies of the vertebræ. If one or more of the bodies of the vertebræ be destroyed by caries, the part of the vertebral column situated above has no longer any firm support, and must sink down; but since the arches of the vertebræ and spinous processes seldom participate in the disease, the vertebral column sinks in its anterior part only, and a curvature forwards occurs, and necessarily also a bulging posteriorly, a so-called *Pott's curvature,* thus named after the English surgeon, Percival Pott, who first described this disease minutely. In every anatomical collection you will find preparations of this unfortunately rather frequent disease. The occurrence of such a forward curvature (kyphosis) of the vertebral column is sometimes the only, but, of course, very certain sign of destruction of the vertebræ.

A second important symptom of destruction of bones are the suppurations which occur in many, and indeed in most cases in the form of *indolent abscesses.* The pus collects low down around

the bone, but does not always remain at the point of its origin, but sometimes spreads further and further; the direction in which this extension occurs will depend upon the greater or less power of resistance of the various soft parts, more rarely upon the laws of gravitation alone. For all the more important disease-nests it is typical and determined by the anatomical conditions. König has recently, by means of investigations very carefully carried out in common with Henke, been very successful in pointing out the courses which these abscesses follow, or rather, for anatomical reasons, must follow for their enlargement and extension. Caries of the vertebral column is one of the most frequent causes of such gravitating or congestive abscesses. Since the disease most frequently commences as chronic periostitis on the anterior surface of the bodies of the vertebræ, it is here that the abscess is first formed; the pus sinks behind the peritoneum along the psoas magnus, and generally appears under Poupart's ligament on its inner side; an extension in other directions, *e.g.* backwards, is also possible, but much more rare. These congestive abscesses are of great importance for diagnosis, and still more for prognosis; they are generally a bad sign. The treatment of them, of which I shall speak later on, is one of the most difficult points in surgical practice. That the pus, obeying the laws of gravity, simply sinks down mechanically—a conclusion to which the term "gravitating abscess" might easily mislead us—is, as I have already remarked, not correct; it spreads most easily in the direction in which there is only loose cellular tissue, and where fasciæ, muscles, and bones offer no resistance, for it is especially an ulcerative process of suppuration going on in a conditionate direction only slightly dependent upon pressure of the pus within the cavity, a kind of enlargement of an abscess such as occurs under other circumstances. If the pus has been formed, for instance, on the anterior surface of the vertebral column, and has found its way in the manner mentioned above to the inner surface of the thigh until it reaches the skin, the latter generally becomes very slowly pierced, not by the mechanical pressure of the pus, but by ulcerative destruction from within outwards, as in the bursting of all abscesses and cavernous ulcers; such a congestive abscess may possibly go on 1½—2 years or more before it bursts spontaneously.

We now come to the *ætiology of ostitis and caries interna*, which we may treat very briefly, because the same causes which lie at the root

of chronic periostitis, and indeed of chronic inflammation generally, also play the chief part here.

It rarely happens, on the whole, that in subjects otherwise healthy a traumatic injury gives rise to the development of a primary chronic ostitis. It may happen, however, that such an affection may become developed in the larger cylindrical bones by violent concussion and compression, with extravasations of blood in the medullary canal, in the form of a chronic osteomyelitis. The same thing may occur also after crushing of the short carpal and tarsal bones. It will, however, always be more common, after such injuries, to find acute processes, *e. g.* acute periostitis, set up. If injuries of the wrist- or ankle-joint are followed by suppuration, the cartilage becomes destroyed, the suppuration spreads to the bones, and fungous ostitis of the small spongy bones, leading to their complete destruction, may follow. Even in perfectly healthy robust individuals, a long-continued traumatic suppuration in a joint may produce a state of anæmia and cachexia, in consequence of which the traumatic inflammation does not run its normal course, but passes into a chronic condition. Scrofulosis and syphilis are the most frequent causes of chronic inflammations in the bones, and in scrofulosis the fungous forms predominate so long as the children are still fleshy and otherwise well nourished. In thin, ill-nourished, anæmic, scrofulous children, on the contrary, ostitis with caseous degeneration not unfrequently becomes developed, as well as the thoroughly atonic forms. The two latter forms are then often combined with partial necrosis. The bodies of the vertebræ, the epiphyses of the joints, the phalanges, and metacarpal bones are the most frequent seats of scrofulous ostitis and periostitis; the jaws and larger cylindrical bones are seldom attacked. In syphilis, ostitis and periostitis osteoplastica occur frequently in the shin bones and in the bones of the skull; caries sicca fungosa occurs in part primarily in the diploë of the cranial bones, in part after periostitis; the sternum, the processus palatinus, and the bones of the nose are often affected; necrosis is frequently combined with syphilitic caries. Many modern writers, *e. g.* R. Volkmann, describe syphilis of the bones under the name of *ostitis gummosa* as something peculiar; I admit that certain combinations occur with especial frequency in it, and that typical forms of disease thus arise, but, anatomically speaking, syphilis in the bones remains always chronic ostitis and periostitis. While, in syphilis, an external agent but rarely sets up the local pro-

cess, this may fairly be regarded as the rule in scrofulosis. Contusions and distortions, slight in themselves, occasion, in scrofulous subjects, disturbances which do not become remedied, but rather extend further and further. In many cases we are unable, even by the closest investigation, to discover local or general causes for the origin of a given case of caries, and I consider it better to admit this at once than to persist in trying to find some questionable explanation by long-continued examination.

I will mention here that the action of the *fumes of phosphorus* upon those employed in the manufactories of lucifer matches produces a suppurative ostitis of the jaws, which generally leads to a form of necrosis which psesents many peculiarities requiring notice in the clinique. *Workers in mother-of-pearl* also sometimes suffer from ostitis, which mostly assumes an osteoplastic form, and is probably caused by the inhalation of the extremely fine dust which fills the rooms in which they work. Gussenbauer is of opinion that this ostitis is of embolic origin, and depends upon the insoluble organic elementary substance of the particles of dust. This disease has hitherto only been observed in Vienna.

LECTURE XXXIII.

Healing process in chronic ostitis, caries, and congestive abscesses. Prognosis. General condition in chronic inflammations of the bones. Secondary swellings of the lymphatic glands. Treatment of chronic ostitis and congestive abscesses. Resections in the continuity of bones.

Before we pass on to the treatment of chronic periostitis, we must add a few remarks concerning the *healing process* in these diseases, and the *prognosis* of them. The former will vary somewhat according to the vitality of the process, as is the case also with ulcers of the skin. If we assume that the inflammatory new growth (formation of granuloma) finally ceases to go on exuberantly, it will then shrink up and become converted into cicatricial tissue. This process will consist, histologically, in a retrograde conversion of the granular tissue into firm, fibrous connective tissue, while its very copiously developed capillary vessels will become for the most part obliterated, and the cells converted into connective tissue and corpuscles of connective tissue. If the caries was combined with open suppuration, the latter gradually ceases and the fistulæ close. If a portion of the bone had already been destroyed and displacements had occurred, the latter no longer become rectified, but the loss of bone first becomes filled up by a cicatrix of connective tissue strongly drawn inwards, and the bones thus displaced become fixed thereby in their false position. This cicatrix of connective tissue generally becomes converted into bone later on. The cicatricial connection of two bones thrust against each other, *e. g.* of two bodies of the vertebræ, which have come into contact in consequence of the destruction of a vertebra lying between them, also becomes bony and the vertebræ thus firmly united. A true compensation, a new growth of bony mass to such an extent that the vertebræ should be raised up again, or that a new bone should be completely or partly

formed, never occurs in caries. If a thoroughly atonic ulcer in bone heals, it may occur in the following manner. Any portions of bone which may have become necrotic must first be thrown off and removed; there must then be formed by the walls of the gap in the bone an active new growth with copious development of vessels, and in the case of larger cavernous ulcers, or of abscesses in the bones, the whole space must be filled up with a granular mass to render its healing possible. These granulations must cicatrise, and afterwards ossify, to render the cure complete. In other cases, the necrotic but not yet separated portion of bone is detached by granulations which spring from the healthy part of the bone behind the diseased, necrotic portion; the torpid process thereby becomes converted into an active, exuberant one, and leads, later on, just as in the former case, to a cicatrix which, if the whole process be completed favorably, eventually becomes ossified. The gaps in the bone, *e. g.* in the centre of a cylindrical bone, are quite incapable of becoming smaller by shrinking, which renders the healing process in the soft parts so much shorter, but must be filled up entirely by new growth. This is the point at which the healing process in ulcers of the bones so often breaks down. The general constitutional conditions which underlie the thoroughly indolent forms of caries are difficult to remove; it is on that account not only difficult to bring the process of ulceration to a standstill, but equally so to effect an active new growth in the diseased parts. If we really succeed in bringing the ulcerative process to a standstill, and if the softened bone reacquires its normal density, fistulæ in the bone not unfrequently remain, which, although painless, continue for many years, and, in many cases, never heal. Such fistulæ in the bones are, however, if the diseased process comes to a standstill, generally innocuous. If you have an opportunity of examining such fistulæ anatomically in macerated bones, you will find that the openings which lead into the bones are lined with an extremely thick, sclerotic layer of bone, exactly as, in old fistulæ of the soft parts, their walls consist of a very hard, cicatricial (callous) mass.

It now remains for me to speak of the healing process in the indolent abscesses of the soft parts arising chronically in these diseases. These abscesses will, in most cases, if they open outwards, not heal until the healing process has commenced in the bone itself. If the cavities of the abscesses are then lined with healthy granulations, which is rarely the case, the walls may no doubt

gradually unite directly with each other. It happens more frequently, however, that such an abscess becomes essentially diminished in size from shrinking of its interior walls, and gradually becomes closed in this manner. For this it is necessary, however, that the process of decay shall have ceased in these walls, and that the tissues shall be amply supplied with blood-vessels. If an indolent abscess does not burst, but remains subcutaneous, while the bone heals, it most frequently happens that a large portion of the pus, the cells of which divide into fine molecules, becomes absorbed, while the interior surface of the abscess becomes converted into a cicatricial tissue which retains the puriform fluid like a fibrous sac. Such sacs containing pus often remain for years in this stage; complete absorption, if only as far as the eventual thickening of the remaining fluid to a cheesy mass, is unfortunately much more rare than could be desired and than is commonly believed. Generally speaking, this whole process of the subcutaneous healing of indolent abscesses by absorption is extremely rare.

In the *prognosis* to be formed for a case of caries, we have first of all to distinguish from each other the fate of the diseased bone and the state to which the whole organism becomes reduced by long-continued suppuration of the bones and soft parts. As regards the fate of the diseased part, we have already spoken sufficiently thereof by explaining, on the one hand, the nature of the destruction and its consequences for the surrounding parts, and, on the other hand, the nature of the possible mode of healing. I will only add here the remark that, in cases of caries of the vertebral column, it is intelligible that the spinal cord may be in danger of becoming involved in the suppuration, or of undergoing a curvature in consequence of the bending of the vertebral column which may interrupt its function: paralysis of the lower extremities, the bladder, or the rectum may occur from caries of the vertebræ. Experience teaches us that this is more rarely the case than might be expected *à priori*, because the spinal cord lies very well defended by the tough dura mater and also bears a pretty high degree of gradual flexion without having its function interfered with. Of general importance for the prognosis is the constitutional condition of the body, *the degree and nature of the febrile reaction*. Chronic diseases of the bones seldom commence with fever, and in many cases, especially when there is no operative interference, and the bursting of the consecutive abscesses is left entirely to nature, the

formed, never occurs in caries. If a thoroughly atonic ulcer in bone heals, it may occur in the following manner. Any portions of bone which may have become necrotic must first be thrown off and removed; there must then be formed by the walls of the gap in the bone an active new growth with copious development of vessels, and in the case of larger cavernous ulcers, or of abscesses in the bones, the whole space must be filled up with a granular mass to render its healing possible. These granulations must cicatrise, and afterwards ossify, to render the cure complete. In other cases, the necrotic but not yet separated portion of bone is detached by granulations which spring from the healthy part of the bone behind the diseased, necrotic portion; the torpid process thereby becomes converted into an active, exuberant one, and leads, later on, just as in the former case, to a cicatrix which, if the whole process be completed favorably, eventually becomes ossified. The gaps in the bone, *e. g.* in the centre of a cylindrical bone, are quite incapable of becoming smaller by shrinking, which renders the healing process in the soft parts so much shorter, but must be filled up entirely by new growth. This is the point at which the healing process in ulcers of the bones so often breaks down. The general constitutional conditions which underlie the thoroughly indolent forms of caries are difficult to remove; it is on that account not only difficult to bring the process of ulceration to a standstill, but equally so to effect an active new growth in the diseased parts. If we really succeed in bringing the ulcerative process to a standstill, and if the softened bone reacquires its normal density, fistulæ in the bone not unfrequently remain, which, although painless, continue for many years, and, in many cases, never heal. Such fistulæ in the bones are, however, if the diseased process comes to a standstill, generally innocuous. If you have an opportunity of examining such fistulæ anatomically in macerated bones, you will find that the openings which lead into the bones are lined with an extremely thick, sclerotic layer of bone, exactly as, in old fistulæ of the soft parts, their walls consist of a very hard, cicatricial (callous) mass.

It now remains for me to speak of the healing process in the indolent abscesses of the soft parts arising chronically in these diseases. These abscesses will, in most cases, if they open outwards, not heal until the healing process has commenced in the bone itself. If the cavities of the abscesses are then lined with healthy granulations, which is rarely the case, the walls may no doubt

gradually unite directly with each other. It happens more frequently, however, that such an abscess becomes essentially diminished in size from shrinking of its interior walls, and gradually becomes closed in this manner. For this it is necessary, however, that the process of decay shall have ceased in these walls, and that the tissues shall be amply supplied with blood-vessels. If an indolent abscess does not burst, but remains subcutaneous, while the bone heals, it most frequently happens that a large portion of the pus, the cells of which divide into fine molecules, becomes absorbed, while the interior surface of the abscess becomes converted into a cicatricial tissue which retains the puriform fluid like a fibrous sac. Such sacs containing pus often remain for years in this stage; complete absorption, if only as far as the eventual thickening of the remaining fluid to a cheesy mass, is unfortunately much more rare than could be desired and than is commonly believed. Generally speaking, this whole process of the subcutaneous healing of indolent abscesses by absorption is extremely rare.

In the *prognosis* to be formed for a case of caries, we have first of all to distinguish from each other the fate of the diseased bone and the state to which the whole organism becomes reduced by long-continued suppuration of the bones and soft parts. As regards the fate of the diseased part, we have already spoken sufficiently thereof by explaining, on the one hand, the nature of the destruction and its consequences for the surrounding parts, and, on the other hand, the nature of the possible mode of healing. I will only add here the remark that, in cases of caries of the vertebral column, it is intelligible that the spinal cord may be in danger of becoming involved in the suppuration, or of undergoing a curvature in consequence of the bending of the vertebral column which may interrupt its function: paralysis of the lower extremities, the bladder, or the rectum may occur from caries of the vertebræ. Experience teaches us that this is more rarely the case than might be expected *à priori*, because the spinal cord lies very well defended by the tough dura mater and also bears a pretty high degree of gradual flexion without having its function interfered with. Of general importance for the prognosis is the constitutional condition of the body, *the degree and nature of the febrile reaction*. Chronic diseases of the bones seldom commence with fever, and in many cases, especially when there is no operative interference, and the bursting of the consecutive abscesses is left entirely to nature, the

patient, with few exceptions, does not become feverish at all. This entirely non-febrile course does not, however, continue long. If the patients have had no fever up to the bursting of the abscess, hectic fever then often sets in, and generally in the form of febris remittens with steep curves, *i.e.* with low morning and rather high evening temperatures, but sometimes also as intermittent pyæmic fever. The earlier the bursting of *large* gravitating abscess is effected and the abscess kept open, so much sooner does the apyretic condition change to a feverish one; a very intense and exhausting febris remittens continua generally sets in; the chronic process of ulceration then not unfrequently passes rapidly into an acute inflammatory process, with a tendency to a diphtheritic condition of the inner walls of the abscess. After the thin, flaky but not ill-smelling pus has been discharged, a serous and afterwards ichorous suppuration sometimes occurs, although of short duration. Pyæmia may supervene at various stages of the disease and terminate in death. It is difficult to explain upon what the unfavorable change after the bursting of congestive abscesses depends, or why the chronic inflammation so rapidly becomes changed into a highly acute one. The common theory is that, from the entry of air into the abscess, a violent inflammation is set up in the walls of the large cavity of the abscess already disposed to decay, and that the oxygen of the air especially promotes decomposition. This theory may be correct for many cases, but not the air alone as such, not the oxygen alone, is the noxious agent, nor yet is it the organic germs also contained in the air which produce the acute inflammation in the walls of the abscess. The further elucidation of these circumstances, which are far from being clear, would lead me too far here. It appears certain that puncturing, or any other mode of opening, is sometimes a sufficient irritation to cause an acutely spreading inflammatory disturbance of nutrition in the generally badly organised walls of the abscess. In many cases, infectious matters may also be communicated by the instruments and dressings; under certain circumstances, the air entering into the abscess may also contain such infectious matters. Concerning the question whether minute organisms only always have about them such infectious matters I have already given you my opinion.

There are still a good many cases in which the suppuration, although profuse, remains healthy, and yet high fever sets in, and the

course then again becomes chronic without violent symptoms. We must not attempt to conceal from ourselves that influences are here present which altogether escape our cognisance. That the chronic process often terminates acutely is an observation which justifies us in the prognosis that the patient's danger is enhanced by the opening of the abscess. I will add here that the organism generally is, essentially, first drawn into sympathy by the *open* suppuration; ostitis granulosa, whether it runs its course as sicca or is accompanied by *slight subcutaneous* suppuration, will, therefore, be less dangerous to life than ostitis atonica with a great tendency to suppuration and to bursting outwardly. This prognostic formula has also its good foundation in the fact that the exuberant inflammatory new growth more frequently occurs under comparatively favorable constitutional conditions, as was remarked above. If the exuberant fungous granulations decay rapidly without external cause, and the suppuration becomes more profuse and thinner, this is a sign that the general nutrition has become bad. The patient's strength is exhausted partly by the formation of the pus, partly by the fever, and is only very deficiently renewed because no normal absorption by the stomach, no true digestion and assimilation takes place; this then reacts upon the local processes, and the general and local conditions stand in the most intimate mutual relation to each other. The smaller the carious nest, the less dangerous it is to the whole organism, but there are certain localities in the body which, independently of the extent of the caries, damage the organism more of themselves than others. Thus suppurations of the vertebræ, with large congestive abscesses, are very dangerous for the organism; caries of the phalanges, even when several of them are affected together, is of much less importance. One great difference in the danger to life consists, namely, in whether one or other of the larger joints is attacked together with the epiphyses; caries of the hip, knee, or foot is much more dangerous than in the shoulder, elbow, or hand. I shall speak further on this subject in connection with the diseases of the joints. Of great importance for the prognosis is also the age of the patient; the younger the individual is, so much more may a cure be hoped for, and *vice versâ*. Every case of caries which occurs after fifty, whether after periostitis or primarily as ostitis, furnishes a very doubtful prognosis for a cure, however unimportant the local process may be at first. I do not remember to have seen caries in old people so frequent anywhere else as in

Zurich. Lastly, the prognosis depends very much upon the constitutional affections which gave rise to the disease. Comparatively the most favorable is syphilitic caries, because syphilis, as such, is most amenable to treatment. Scrofulous caries in well-nourished children is also seldom dangerous to life, because scrofulosis either yields to appropriate treatment or dies out spontaneously. But caries in scrofulous and at the same time atrophic children is dangerous, for such children readily die of exhaustion. The most unfavorable is the prognosis of caries with already existing tuberculosis; it very seldom becomes healed, the pulmonary tuberculosis generally makes rapid progress, acute miliary tuberculosis of the serous membranes occasionally supervenes and soon terminates fatally.

As regards the history of patients sinking slowly in consequence of chronic inflammations, such patients gradually become more and more emaciated, pale, extremely anæmic, and generally have at last œdema of the lower extremities, with steadily increasing loss of appetite, and die from marasmus, after suffering for years, often in a frightfully slow manner—sometimes falling asleep quite quietly, sometimes struggling for days with death. It was formerly generally assumed that death here resulted from gradual exhaustion alone; more careful post-mortem examinations have shown, however, that the exhaustion and the constantly increasing deficiency of formation of blood often have very recognisable causes. In these subjects, namely, we very frequently find the liver, spleen, and kidneys in a state of *lardaceous*, or *amyloid degeneration* (*hyalinosis*, O. Weber), a kind of morbid change which consists therein that a peculiar product is secreted by the smaller arteries within the substance of those organs, which is distinguished on the one hand by the lardaceous appearance and consistence which it produces in the diseased organs, on the other by its reaction; on the addition of iodine or sulphuric acid, namely, this product assumes partly a deep reddish-brown, partly a dirty violet-brown colour, with change of colour to green and pale red. Concerning the nature of these products various opinions exist with which you will become more familiar in connection with pathological anatomy. I will only remark here that this reaction with iodine and sulphuric acid resembles that of cholesterine, and that Heinrich Meckel, of Hemsbach, therefore believed that the lardaceous matter owes its reaction to the large amount of cholesterine it contains. Others have

thought that the product in question is related to starch, and Virchow, who adopted this view, therefore called it amyloid. Kühne has shown that both views are untenable; the so-called amyloid is a peculiar body nearly related to albumen; it is distinguished from the latter especially by the circumstance that it is insoluble in acids which contain pepsine. This body is at least very interesting and remarkable on account of the mode of its appearance; it and fibrine are the only organic bodies known to us which penetrate the walls of the vessels in a fluid state, and then acquire a solid consistence outside of them without any apparent necessity for the energetic action of cells, as in the formation of the tissues.

The saturation of the liver, spleen, and kidneys, as well as of the walls of the arteries of the intestinal canal and of the lymphatic glands, with lardaceous matter, must naturally exert a great influence upon the formation of blood, and ultimately check it altogether. Extensive chronic suppurations dispose in a high degree to lardaceous disease; this is, therefore, greatly to be feared in patients suffering from extensive caries, and is, unfortunately, in many cases not preventible. In addition to tuberculosis and lardaceous disease, which are but too frequently combined with each other, these poor patients are sometimes also threatened with the usual forms of acute and chronic diffused nephritis (morbus Brightii), sometimes running an acute, sometimes a chronic, course.

I will mention further that in chronic inflammations of the periosteum and bones especially, the nearest-seated *lymphatic glands* very often become implicated. As, in the acute inflammations, the lymphatic glands so frequently become infected, and also acutely inflamed, by products which reach them from the inflamed parts, so does the same thing occur from similar causes in chronic inflammations. The lymphatic glands swell gradually and painlessly, but in the course of months and years very considerably. The tissue of their walls becomes thickened, individual lymphatic ducts obliterated, and others perhaps dilated; the process seldom goes beyond this hyperplastic swelling, but sometimes small abscesses and cheesy nests are formed.

It is now time, after having considered chronic periostitis and ostitis from all points, that we should also think of their *treatment*. Here again, after having spoken of those diseases in their most varied extension and combination, we must begin with simple

chronic periostitis. The treatment must be at once local and general, and, in all the cases in which dyscrasic causes are demonstrable, must be especially directed to them. In respect to this point, I must refer you to what I said when speaking generally of these dyscrasias in the chapter on chronic inflammation. We shall here, therefore, have to occupy ourselves especially with the *local remedies*. Rest of the diseased portions of the body is to be recommended as a first most general rule for the treatment of chronic inflammation of the bones, for movement, blows, a fall, or the like accidental injuries, may convert a mild, perhaps favorable course into an acute, dangerous one. For diseases of the bones of the lower extremities, therefore, rest in bed is, in most cases, one of the first conditions; for the upper extremities, rest in a sling. This rest is of especial importance in diseases of the bones in the vicinity of the joints, in which it often comes of itself, because movements cause too much pain. Many forms of fistulous caries in the diaphyses of the smaller and larger cylindrical bones no doubt run, when the escape of the pus outwards is fully established, into a stage so painless and free from irritation that movement does not affect the diseased bone, and in such cases a moderate amount of motion may be permitted. A raised position of the inflamed part is a good auxiliary means for the cure, since it prevents interruption to the venous circulation, and this aid to the return of the venous blood must not be under-estimated. Shampooing may, in these earlier stages, be tried cautiously.

In practice you will not readily be able to fulfil these first therapeutical requirements; you will find especially that grown-up people go about with and upon their diseased limbs as long as they can, *i.e.* as long as the pain is not too severe. Since you cannot promise the patient to cure his disease with certainty if he remains some weeks in bed, because such cases go on frequently with the most careful treatment for months and years, he will follow his occupations as long as possible. If your patient is so situated that the existence of a family depends upon his daily labour, his position is an especially unfavorable one. In the case of children it is equally difficult to keep them constantly at rest; a grown-up person must watch them all day. This is impossible, not only in poor families, but equally so in families in tolerably good circumstances if there are many children. Represent to yourselves the state of things in such a case! It is easy to give directions: the child must remain constantly recumbent for several months, but be wheeled cautiously

into the open air every day, or lie in the shade in the garden in favorable weather. This, if it must be carried out for some years, costs a great deal of money, besides which such a child engrosses almost the whole attention of a careful adult nurse. It is just this daily, even hourly, observance of the dietetic and hygienic requirements of a child thus affected chronically which calls for unusual endurance and intelligence. Much more readily are sacrifices made for expensive medicines, or for a sojourn at a watering-place, &c., "to put an end to the matter," as fathers of families are apt to express it when they have become tired of the long daily trouble with the sick child and the disturbance in the household and consequent loss of their domestic comforts. We must take the general circumstances into consideration in such cases, that we may do the best we can, and order mechanic support which shall prevent the weight of the body from acting upon the diseased bone. I have afforded you this preliminary glimpse at your future practice that you may not be too much disappointed later on. You will frequently arrive at the conviction that there are many, especially chronic, diseases which are by no means incurable, but which, for social reasons, are scarcely ever cured.

If the first symptoms of a chronic periostitis and ostitis present themselves, the treatment must be directed to the dispersion of the infiltrations. Strictly antiphlogistic means are of little use for this purpose. The application of leeches or cupping-glasses, the internal administration of purgatives, the use of bladders filled with ice, are, in my opinion, remedies which are efficacious only in the acute exacerbations of chronic inflammations; their effect always passes off rapidly, and the use of local abstractions of blood and purgatives, if repeated frequently, may even have a deleterious influence. The leeches and cupping-glasses applied time after time cause local irritation, and finally render the patient anæmic, and continued purging exhausts his powers; we must, therefore, employ these remedies sparingly, and reserve them for the more acute exacerbations of the process. The constant application of bladders with ice has recently been very strongly recommended by Esmarch in chronic inflammation. In cases *accompanied by violent pain* I have seen *very good results* from this treatment; in other cases I can see no clear indication for it.

The absorbent and milder derivative remedies are those which most frequently come into use in the first commencements of chronic

inflammations of the bones—the officinal tincture of iodine, iodide of potassium ointment, mercurial ointment somewhat diluted, mercurial plaster, ointments with a concentrated solution of nitrate of silver, hydropathic wrappings, light compressing dressings, shampooing. With these remedies and the appropriate hygienic directions, and perhaps a visit to one of the watering-places formerly mentioned in connection with scrofulosis, we generally begin the campaign against the diseases of which we have been speaking, so long as they are in their early stage, and sometimes succeed in arresting the process at an early period of their development. The retrograde metamorphoses occur in the early stages either without leaving behind them any trace of morbid change, or perhaps with a moderate thickening of the bone by osteophytes, the absorption of which can no longer be effected. The most successful is the treatment of the syphilitic diseases of the bones at this stage by an energetic course of anti-syphilitic remedies. If the process goes on, and the caries runs its course without suppuration, we continue the treatment, to which, in individuals who appear able to bear it, we may add the derivative remedies which act more powerfully upon the skin. If the signs of suppuration appear, and abscesses form, you may still go on for a time with the absorbent remedies in the hope of effecting the absorption of the pus; this will, indeed, not happen in the majority of cases, and the question will soon force itself upon us—shall we open the abscess artificially, or shall we wait until it bursts spontaneously? Concerning this point, I give you the following general rules:—*If the abscesses spring from bones in which any operative interference is either impossible or undesirable, e.g.* from the vertebræ, the sacrum, the pelvis, the ribs, the knee-joint, &c., *do not interfere* but rejoice, in the interest of your patient, over every day that it remains closed, wait quietly until the opening takes place of itself, because in this way symptoms comparatively the least dangerous will present themselves. If I have not always adhered to this principle I have never failed to regret it, and it gave me great pleasure to read that Pirogoff has expressed himself on the subject in almost the same words. Experience has shown fully that all our operative manœuvres which have for their object an imitation of the gradual spontaneous bursting of these abscesses do not act so favorably as the gradual penetration of the skin from within outwards by a process of ulceration. Various methods have been proposed, especially for the opening of large congestive abscesses, according to the views adopted as a start-

ing-point. It was believed for some time that the pus must flow out slowly to prevent inflammation of the walls of the abscess. To attain this, *setons* were introduced and the pus allowed to dribble away at the openings formed by the needle. It was then thought that, in addition to this slow escape of the pus, the skin must be slowly pierced, and some caustic was applied to the thinnest parts of the abscess to form an eschar, which came away slowly, after which the pus flowed out gradually. Later on it was believed that the entrance of air must at all events be prevented, because this was the dangerous element in the whole matter. A trocar was introduced, a part only of the pus very cautiously allowed to flow out, and the opening then closed exactly, or Abernethy's so-called subcutaneous puncture was made, *i.e.* the operator took a thin sharp knife, pushed the skin over the sac of the abscess forcibly upwards, introduced the knife, and allowed a large portion of the pus to flow out; then drew out the knife rapidly, and let the skin slip back into its natural position, so that the puncture in the skin did not communicate directly with the opening into the abscess, but the latter was covered by the skin; the opening in the skin was then carefully closed. Guerin advised that the pus should be drawn out through the canula of a trocar with a syringe, a proceeding which has again come into use quite recently with an improved apparatus under the name "aspiration pneumatique sous-cutanée" (Dieulafoy). After this, great importance was attached to checking the suppuration in the walls of the abscess; it was thought that this might be effected by injecting a solution of iodine after the pus had been drawn off. This method was much approved of in France. A French surgeon, Chassaignac, recurred with great enthusiasm to the use of setons, but in the form of thin india-rubber tubes with perforated walls, which greatly facilitated the flowing out of the pus (drainage). The Scotch surgeon Lister attaches especial importance to the disinfection with carbolic acid of all the instruments and dressing materials to be used for opening and dressing these abscesses, and to the careful avoidance of the access of air. His method, like all the earlier ones, has found enthusiastic admirers. It is not very easy to form any clear notion as to the importance of all these therapeutic methods, but you may almost always conclude from so great a number of remedies and methods recommended, that diseases are then in question which are very difficult to cure, and that no one of these methods is applicable to all cases. We will

examine briefly the methods just mentioned. Withdrawal of the pus all at once, in whatever manner we may undertake it, (except by free incisions into congestive abscesses, which are applicable to very few cases only), has at first a pretty favorable result if carried out slowly and cautiously, whether by means of a trocar or subcutanously with a knife, with or without Lister's carbolic paste. If the opening is well closed and heals, there is generally no fever afterwards, but the abscess fills again with striking rapidity. An abscess which required, perhaps, ten months for its first formation may become completely filled again in ten days. A fresh puncture is made and the opening again heals, but the patient begins to have slight fever; the pus again collects rapidly. A third, fourth, or even fifth puncture is made, always at a fresh point; the patient already has more fever, the abscess has become steadily hotter, and therefore painful, and the patient looks weak and pulled down. The punctures now no longer heal, the earlier ones probably break open again, there is a continuous escape of pus in spite of every care; air sometimes enters the abscess, especially if its walls are rigid. A fistula now forms perhaps, there is constant fever, and the further course of the disease is, as I described to you on a former occasion, for the most part an unfavorable one. If, after the puncture, you inject iodine, this will not, according to my experience, essentially affect the course of things, although in a few cases I have seen rapid healing of indolent subcutaneous abscesses occur. Not very different is it if you effect the opening of the abscess and discharge of the pus by means of setons, drainage, caustics, or the syringe; I have not seen results from any of these methods which at all justified the recommendations of their proposers. If the patient is suffering much from the growth of the abscess, from pain and a feeling of tension, &c., you may draw off some pus to satisfy him with the Dieulafoy syringe; this will do comparatively the least harm.

The unfavorable course just described may indeed be just the same, under certain circumstances, if you do not interfere with the abscess at all, but leave it to itself and wait for it to burst spontaneously; but all then goes on less violently and more slowly, and fever sets in later. Recoveries are, no doubt, observed with all the methods named; more cures, I believe, but certainly fewer fatal terminations, occur with the expectant treatment. I am convinced that where, after iodine injections, drainage, &c., a recovery took place, it would also have occurred if the natural course had not been

artificially interrupted. Any actual proof that a given case would have run such and such a course if this or that thing had not happened is, of course, quite out of the question. If I sum up the cases I have had the opportunity of collecting in my clinical and consultation practice, I can assure you that amongst a very large number of cases in which congestive abscesses of the vertebral column were skilfully opened, for the most part by other surgeons than myself, I know of very few only in which the course was a favorable one; all the others terminated fatally all the more quickly. I must, therefore, recur to the principle laid down above, that abscesses of this kind, especially congestive abscesses with caries of the vertebral column, are a *noli me tangere*. It is, indeed, often very difficult to go on waiting in such cases; patients grow impatient, especially in private practice; the surgeon is urged to do something, and is blamed for undertaking nothing; the public believes once for all that if the pus were but entirely removed recovery must follow. The surgeon also at last becomes impatient; it is painful to see how the abscess goes on enlarging from week to week, from month to month; every local and general remedy has been exhausted, the surgeon finally deviates from his principles, the opening is made; at first all appears to go well, but the rejoicing is of short duration; how things go on afterwards you know already. You may do what you will in such cases, the public will always blame you for the generally unfavorable result.

Somewhat different is the state of things in the case of *smaller abscesses* which result from *disease of the bones of the extremities*, or of larger indolent abscesses which lie above the fasciæ and have no connection with diseased bone. In those suppurations which communicate with the larger joints we also gladly put off making an opening, for reasons which will be given later on when speaking of diseases of the joints. In indolent abscesses about the diaphyses, waiting is not of much use; I there consider early opening as admissible, except in the case of syphilitic gummy tumours, in which, even in the stage of distinct fluctuation, absorption may still take place, and in evidently tuberculous subjects or very weakly individuals, in whom no operative interference is indicated. The opening of such abscesses would only cause increased suppuration without doing any good. In all other such cases I am in favour of opening the abscess, and that by a free incision, so as to gain a clear notion of the nature and extent of the process; the reaction which takes

place under these circumstances is slight, there is often no fever afterwards, often only slight fever, lasting but a short time. Let us assume a case of chronic periostitis with caries superficialis in the diaphysis of a cylindrical bone: an abscess formed, and was opened; the wound was covered with lint at first, and we waited to see what appearance the ulcerative surface would present. According as the ulcer shows more tendency to exuberant granulation or to decay, we must modify the local treatment, and I should only be repeating myself here if I were to refer again to the remedies to be employed. The treatment should be aided by local baths. Hydropathic wrappings, cataplasms, lint dressings moistened with various fluids, serve as the dressing. It will become more and more evident, in the further course of our observation, to what extent the disease in the bone depends upon the general condition of the patient. If you have to deal with a miserable, tuberculous individual, all local remedies will be useless; if the general condition be good, you may fairly entertain the idea of an energetic local treatment—daily painting of the ulcer with tincture of iodine, the application of red precipitate ointment, frequent and active cauterisation with nitrate of silver, dressing with diluted liq. ferri sesquichlor. In other cases we abandon entirely all idea of aiding the spontaneous healing, and remove the whole of the diseased portion of bone. For this purpose there are different kinds of cutting bone-forceps and saws of the most varied form; *I prefer the scraping off of the diseased portion of bone with sharp spoons to all other modes of proceeding.* If the ulcer in the bone has been cleanly removed as far as the sound parts by any of these means, and the general constitutional condition is tolerably good, we may hope that the wound in the bone thus made will heal normally by healthy granulation and suppuration like other wounds of bone. In fairly strong subjects we may venture much; I have sometimes laid open the whole abscess, scraped it out thoroughly, united the skin by means of sutures, and drained the cavity of the wound freely. Sometimes rapid union of the whole cavity ensued.

If it is a question of an ostitis interna, a caries centralis of a cylindrical bone, or of one of the larger spongy bones, such as the calcaneus, there may, under certain circumstances, be an indication, if an abscess in the bone has gradually made itself recognisable by very violent pain, or by some of the other symptoms mentioned already, to chisel away part of the bone or otherwise lay open the

cavity in it to allow the pus to escape. I recommend this method to you, however, only in those cases in which you are certain about the diagnosis, for we do our patient no slight injury if we lay open a sound medullary canal. Very acute osteomyelitis, with its frequently dangerous consequences, may be the result of such an operation, while a similar injury to a diseased bone is not usually followed by any severe symptoms. In other cases you may wait for the spontaneous opening of the abscess through the bony walls; you can then examine it with a probe and judge clearly of the case. What are the difficulties which interfere with the healing of such cavities in the bones I have already told you. If the process remain for a long time in the same stage it may be advisable to enlarge the opening in the bone, to lay the cavernous ulcer open, and to remove its walls. Such an opening of the cavity of the abscess becomes the more necessary if small necrotic portions of bone lie in it and prevent it from healing, *i.e.* if the caries is a necrotic one. But all these manipulations are indicated only so long as the constitutional condition is still good; if pronounced tuberculosis or extreme marasmus exist, when a fatal course is in every case to be expected, no surgeon will think of undertaking operations whose results can only be favorable if the local transformation of the new wound in the bone may run a normal course. These operations, which we may class, partly at least, with *partial resections in the continuity*, have lost their cruel and terrible element since chloroform came into use, thanks to which patients are no longer conscious of the scraping, chiselling, hammering, and sawing going on in the bone.

In those cases in which the caries has extended so widely that it involves the whole thickness of a cylindrical bone at one point, we might propose to saw out the whole diseased portion in the entire thickness of the bone. A case of this kind is, in the first place, very rare, and, secondly, the success of such an operation is extremely doubtful. Out of the middle of the fibula, the radius or ulna, the metacarpal or metatarsal bones, we may no doubt saw a piece entirely without interfering much with the function of the extremity. If we adopted the same plan for the humerus, the femur, or the tibia, and the healing process really became completed, the functions of the extremity would be performed very incompletely, and would have to be aided by splints. For the lower extremities a wooden leg would be more serviceable than a foot the

continuity of the bone of which is interrupted for a considerable space. It has been believed that in such cases the periosteum detached from the bone before the operation and left in the wound would form new bone, but after operations for caries of the diaphyses the reproduction of bone has been very scanty, so that not much reliance can be placed upon it.

Lastly, as regards those cases in which a cylindrical bone is diseased throughout from periostitis, external and internal caries, and partial internal and external necrosis, cases which, on the whole, are rare, it would be a question only of *extirpation of the whole bone*, or of *amputation of the limb*. Cases in which the whole ulna or radius has been extirpated have sometimes terminated favorably, an instance of which I communicated to you formerly from my own practice; the extirpation of the whole of the first metatarsal bone, &c., has frequently been performed successfully. I know a case also in which the whole humerus, with the exception of the thickened periosteum, was taken out; but the patient died some months after the operation from an internal disease—if I mistake not, from Bright's disease—so that we could not judge how far the limb might have been useful. The hand might have performed its functions in spite of the absence of the humerus, and that would always have been a great advantage for the patient. Caries of the short spongy bones and of the epiphyses of the joints is always so closely connected with diseases of the joints that we cannot speak of it until later on.

The treatment of the general marasmus which finally shows itself in diseases of the joints with extensive suppurations is to be treated on general therapeutic principles. It must be our endeavour either to prevent the setting in of this dreaded state of things or to put it off as long as possible. It is the surgeon's duty under all circumstances to keep his patient alive as long as possible. It is his duty also, therefore, even when a patient's case is almost entirely hopeless, to do all that is possible to keep up his strength. Nutritious diet must be given as soon as any signs of emaciation and deficient nutrition are observable. Later on, it is useless. Unfortunately, it is not in our power, even by the most careful regulation of the diet on physiological principles, to keep the muscles, nerves, and blood of individuals thus suffering from marasmus in a favorable condition. If the organism is to become stronger and stronger by means of nutritious diet it must be capable of assimi-

lating the food provided for it, its digestive process must be regular, its chyliferous vessels and the muscles of its intestines must perform their functions actively. If the food thus really enters the blood normally it still depends upon the energy of the circulation and the activity of the tissues whether they take up and assimilate what is provided for them, or merely let it pass through them. Lastly, the effete matters injurious to nutrition must be removed normally. The human organism is an extremely complicated machine, which is not only kept in motion by means of fuel, but must also obtain its own substance, repair damaged parts, and grease its wheels and springs by the aid of this fuel. We can make a sick or a healthy man weak and finally let him die of starvation by depriving him of food, but we cannot make either a sick or a healthy man fat at will.

In the case of children and young people, a young surgeon may easily be deceived as to their powers, and you will learn amply from experience that wretched subjects, reduced to skeletons and anæmic in the highest degree, improve wonderfully and unexpectedly if the diseased limb, which appeared to be consuming their lives, is amputated; for that, under such circumstances, success is *rarely* to be hoped for from resections, is evident. How fully we may here carry out the principle of conservation of the limbs by sawing out the diseased portions of bone can only be prognosticated in an individual case, and then merely with approximate certainty.

LECTURE XXXIV.

NECROSIS.—*Ætiology. Anatomical conditions of Necrosis totalis and partialis. Symptomatology and diagnosis. Treatment. Sequestrotomy.*

GENTLEMEN,—We have already spoken repeatedly of "*necrosis,*" and you are aware that we understand thereby gangrene of the bones, the death of a bone or part of a bone. I have also already told you that the dead bone is called *sequestrum*. You are further already aware that necrosis may occur either as a consequence of acute processes, or in combination with processes of ulceration, as "*caries necrotica.*"

As in the death of any part of the body, the cessation of the circulation is the immediate cause of the necrosis also, while the cessation of nervous action is not followed by this process, although an interruption of nutrition, an atrophy of the bones in paralysed parts, is sometimes observed. Necrosis may depend mediately upon various circumstances. We will group these briefly together here.

1. *Traumatic influences.*—These include violent concussion and compression of the bone, with or without an external wound. The course of things is the following:—In consequence of the injury, extravasations occur in the medullary canal, in the spongy bones, and perhaps also in the compact bony substance, sometimes also beneath the periosteum. If these lacerations of vessels are so extensive that their consequences are not obviated by the collateral circulation difficult of establishment in the bone, a part of the bone will receive no more blood; it will die, and there may ensue, according to circumstances, a necrosis centralis, or superficialis, or totalis (the latter most readily in small bones). The piece of dead bone lies as a foreign body in the organism, but is still in continuity

with the sound portion of bone. How the separation of the sequestrum by softening of the bony substance at the boundary of the living bone occurs has been explained already. Another form of injury is the laying bare of the surface of the bone, or the sawing through of a bone, where the sawn surface becomes surface of bone. In compound fractures, a portion of bone may become so much separated from the soft parts, and its circulation thereby so completely cut off, that it becomes necrotic. That the exposed bone does not always become necrotic, any more than the sawn surfaces of the bone, that the bone, just like the soft parts, is capable rather of forming granulations directly, has also been explained formerly. Nevertheless, superficial and partial necrosis not unfrequently result from such causes, because either extensive coagulations take place in the extremities of the injured vessels of the bone, or the vessels become compressed in very acute suppuration within the Haversian canals, and are destroyed thereby.

2. *Acute peritositis* and *ostitis*, as well as *osteomyelitis*, are very frequent causes of extensive and especially of total necrosis of long cylindrical bones. In suppuration of the periosteum the supply of blood from those vessels which enter the bone from it is cut off; the suppuration also extends in and through the Haversian canals as far as the medulla. If the latter suppurates, necrosis is inevitable and will extend as far as the inflammatory process has done. Exactly the same result will follow in primary acute ostitis and osteomyelitis, with secondary periostitis.

3. *Chronic ostitis* and *periostitis* may be combined with necrosis, since, in strict analogy with the acute process, suppuration, or decay of the inflammatory new growth, with formation of detritus or caseous degeneration, extends into the bone, and the circulation in the latter becomes so much interrupted that a part of the bone no longer receives any nourishment, and must, therefore, become necrosed. The atonic forms of caries lead more readily to necrosis than the fungous, as has also been mentioned formerly.

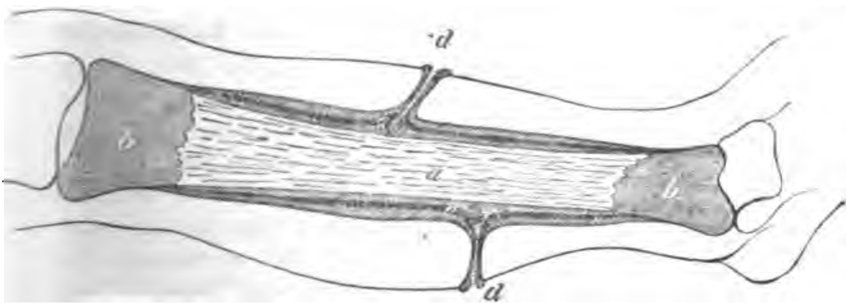
Of more importance theoretically than practically is the necrosis which is regarded as resulting from thrombosis or embolism of the main branch of an arteria nutritia ossis. From post-mortem examinations in the human subject this form of necrosis has, as yet, scarcely been clearly demonstrated; it is also highly improbable, because the arterial supply in the adult bone takes place from so many sides that the obstruction of one of the many communicating

branches does not suffice to interrupt completely the circulation in any considerable portion of the bone. If the collateral circulation in the bone is, for mechanical reasons, rendered inactive from dilatation of the vessels, and there is danger of partial necroses from extensive stases in the capillaries, as was clearly pointed out formerly, the connection, arrangement, and uniform distribution of the capillaries in the dense cortical substance suffice, if the supply of blood be interrupted from the one side, to effect it from the other. There do not exist in the bone any such sharply defined nets and groups of capillary vessels as, for instance, in the skin, but all the capillaries are in intimate continuous connection in all directions, as also in the muscles. Experiments of this kind have, indeed, been made in the rabbit by stopping up with a small pin the foramen nutritium in the upper part of the tibia, when partial necrosis was observed to occur around the pin. I have imitated these experiments, and obtained the same result if I drove the pin into any other part of the bone, and believe, therefore, that the necrosis thus caused artificially finds its explanation simply in the particular kind of injury to the bone.

We shall do well now to examine more closely into the anatomical progress of the necrosis, especially of that which occurs after acute periostitis and osteomyelitis. I have already told you on various occasions, as well in reference to the healing process of fractures as to chronic ostitis and periostitis, that the circumference of such suppuration-nests almost always sympathises in such a manner that osteophytes form upon and in the bone, in the development of which the periosteum (after fractures, also the surrounding parts) play a very essential part. While, after fractures, this new formation of bone brings about the solid reunion under the name of "callus," in chronic ostitis and periostitis it is rather an incidental product of irritation, which acquires no further importance subsequently. The same holds good for cases of superficial necrosis. If around the seat of the disease, whether during the exfoliation of a flat bone of the skull or during the formation of sequestra on a sawn surface, the bone becomes thickened by fresh deposits of osteophytes in the neighbourhood of the sequestrum, this is of no further practical consequence. The state of things differs in compound fractures. If necrosis of broken ends or of fragments of bone, for the most part detached, occur there, the new formation of bone taking place in the vicinity not only produces

future firmness of the bone, but it may also easily happen that the sequestrum may be completely surrounded by the exuberant bony new growth, and have to be removed in part artificially. *But this new formation of bone attains its greatest importance in total necrosis of entire diaphyses. It is destined to replace the bone which becomes lost.* We must now examine more closely into this process, so extremely important and so wonderfully arranged by nature. We start from an acute, total periostitis and osteomyelitis, with necrosis of the diaphysis of a tibia perhaps. The whole periosteum and the medulla have been destroyed by suppuration. In the interior of the bone the pus breaks down into detritus, or becomes decomposed; the periosteal suppuration has penetrated the skin from within outwards at several points, and the circulation has ceased in the diseased diaphysis; *the whole diaphysis forms a sequestrum;* a longitudinal section reveals the following state of things (Fig. 90):

FIG. 90.



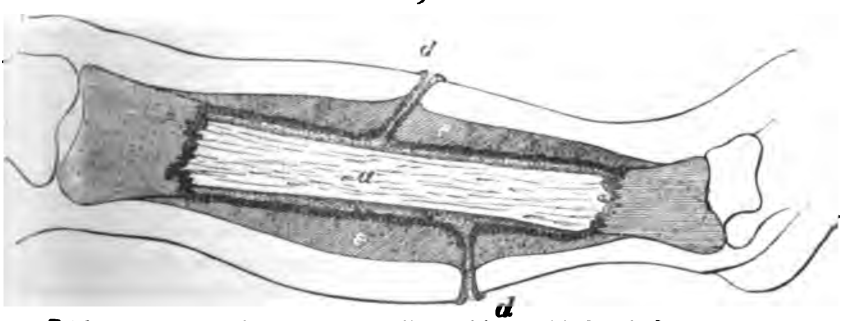
Total necrosis of the diaphysis of the tibia. Diagrammatic drawing.

a, the bony sequestrum; *b, b*, the upper and lower boundary thereof; *c, c*, suppuration which surrounds the sequestrum, and has broken through and runs off at *d, d*. The darkest layer, *e, e*, is the wall of the large suppurating cavity, which consists of tissues infiltrated plastically (connective tissue, tendinous tissue, also muscle perhaps), and on its inner surface, which is turned towards the sequestrum, is covered, like every suppurating cavity, with a layer of granulations which constantly produce fresh pus. I will mention here that this description, as well as that representing the state of things in acute peritonitis, differs somewhat from that of other surgeons and anatomists therein that they assume that the tendinous portion of the periosteum is raised up from the bone by the pus in the form of a bladder. This is incorrect, because the

tendinous portion of the periosteum is not so elastic as to admit of being raised up rapidly like a blister of the epidermis, and this raising up could not occur at those points where the periosteum is wanting, *i.e.* where tendons are attached to the bone. The inflammation and suppuration begin partly *from the surface of the bone, partly in the softer portion of the periosteum, in its external layer.* The tendinous layer takes little part therein, but rather undergoes decay for the most part. I have met with very striking post-mortem examples of this. Those anatomists and surgeons who believe in a raising-up of the periosteum consequently regard the darker layer, *e, e,* as infiltrated, thickened periosteum. This is only correct conditionally. It may happen that a portion of the periosteum does not suppurate, and forms a part of this layer, but other surrounding parts may become so thoroughly indurated by plastic infiltration as to form a dense abscess-membrane, as we see not unfrequently in abscesses of the soft parts. Any one who adheres to the notion of the exclusive capability of the periosteum to form bone will, for theoretical reasons, recognise only thickened periosteum in this layer, *e, e,* in which eventually the formation of the bone takes place. But we have already seen, in the formation of callus after fractures, that in other soft parts also which lie near bone new bone is formed, under certain circumstances, in considerable quantity, and are therefore not compelled to recognise in this thickened layer only thickened periosteum. But we are going too fast! Let us return to the example before us. The suppurating cavity around the sequestrum cannot become closed until the sequestrum has been removed, but the latter is still attached at both ends. How the separation is effected you know already: at *b, b,* at the edge of the living bone, an exuberant interstitial granulation occurs, by means of which the bone here becomes destroyed for a short distance, so that the bony substance at these limits is entirely replaced finally by a mass of soft granulations, and the separation of the sequestrum thus effected. The granulations here formed also break down to a certain extent, and suppurate, and the sequestrum lies loose in the suppurating cavity, which is lined throughout with exuberant granulations. This separation of the sequestrum requires a long time in the thick cylindrical bones, generally several months, sometimes more than a year. During that time the pus continued to escape at the points at which the external openings in the skin were formed. If you

pass a probe through these openings you will always feel during the whole time for the most part the smooth surface of the diaphysis. During this process of separation of the sequestrum something has, however, occurred in the immediate neighbourhood, to which we must now direct our attention. In the thickened layer of the suppurating cavity *e*, *e*, namely, fresh bone has been formed, and that everywhere uniformly round about the sequestrum and corresponding to its length. At the point where the thickened layer joins the periosteum of the epiphyses and the capsule of the joint, the newly formed bone has extended so far that the capsule of bone above and below is in intimate connection with the epiphyses. The longer the sequestrum remains in the cavity, so much more does the capsule of bone increase in thickness. It acquires in time a considerable degree of strength, and may, in the course of years, if the sequestrum does not come away in the mean time, have attained a thickness of $\frac{2}{5}$ inch, and consists at first more of a porous bony mass, but later on acquires great density. Around the sequestrum, therefore, a complete mould has been formed, such as we make with plaster-of-Paris when we wish to take a cast of a body, but the bony mould has some openings, namely, where the pus escapes. These do not close up, because they are prevented from doing so by the constant passage of the pus. The last drawing (Fig. 90) has now assumed the following appearance:

FIG. 91.



Total necrosis of the diaphysis of a cylindrical bone with detached sequestrum and newly formed bony case. Diagrammatic drawing.

The sequestrum *a* is detached and surrounded by pus secreted by the granulations mentioned already; *d*, *d* are the fistulæ which lead to the suppurating cavity, and which have received the name of *cloacæ*; *e*, *e* is the bony capsule resulting from the ossification of

the thickened walls of the abscess, the so-called bony case. The thickening of the latter would continue steadily if the irritation caused by the sequestrum also continued. If you now assume that the sequestrum has been removed from its cage, of which we shall say more hereafter, you see that, although the bone is deprived of the whole of its diaphysis, no interruption of its continuity exists, because the newly formed bony capsule replaces the portion of bone removed.

FIG. 92.

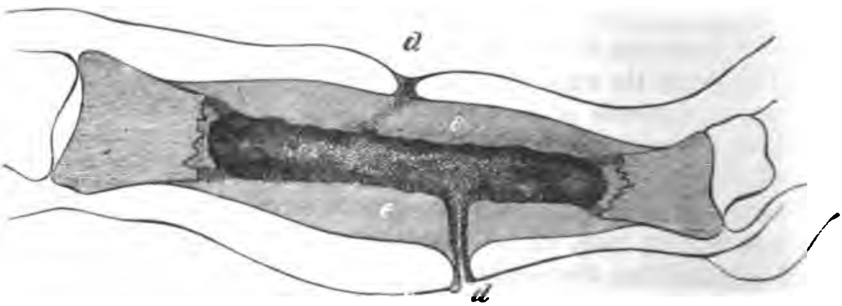
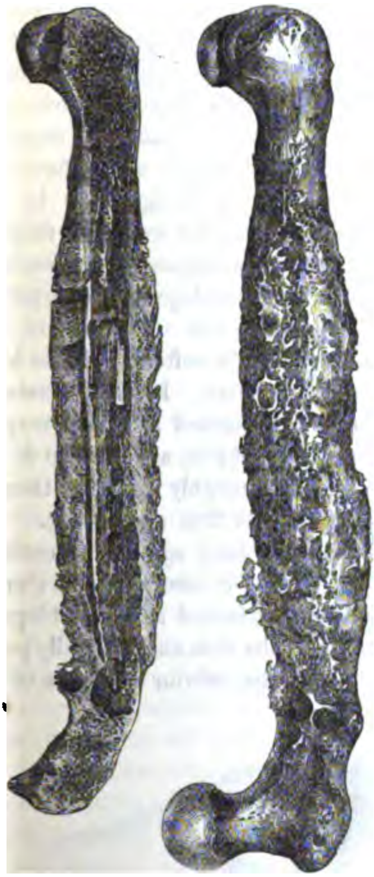


Fig. 91 after the removal of the sequestrum.

But what happens now? Does the cavity in which the sequestrum lay continue to suppurate? No; if everything goes on normally this cavity becomes filled with granulations like similar bony cavities in central caries; these granulations become ossified, and the bone is completely restored, at least as far as the form is concerned. Whether in such cases a new medullary canal is formed, as after the cure of fractures, observations are wanting to determine. According to analogy, however, this is not improbable. The healing up of these cavities after the removal of the sequestrum often requires months or years, and sometimes does not take place at all, especially if the respective individuals were weak generally, or became so from the long-continued suppuration which accompanies the whole process. Albuminuria not unfrequently developes itself in these long-continued suppurations of the bones, although mostly in a mild form. Whether it may disappear spontaneously after the healing of the bony cavity I do not know; it would be interesting and important for prognosis to collect information on this point. If the sequestrum has been removed, the thickening of the bony capsule ceases, and the process of ossification is now set

up in the cavity filled with granulations. What I have shown you here in diagrammatic drawings you now see in these beautiful preparations of the anatomical and surgical collection of Zurich (Figs. 93 and 94).

FIG. 93.



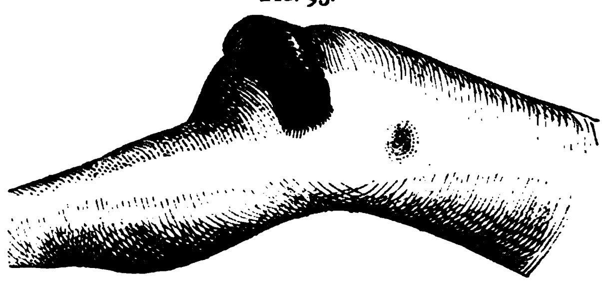
a. Total necrosis of the diaphysis of the femur, with considerable bony case, by which the dead piece of bone is replaced; through this bony case pass several rather large openings inwards to the sequestrum. *b*. The same preparation in a longitudinal section.

FIG. 94.

a. Tibia of a young man after total necrosis of the diaphysis; about two years previously, I had removed the sequestrum *b*. The cavity has become almost completely filled with osteophytes. The patient died of carbuncle.

You know the usual normal course of a case of total necrosis. I must make you acquainted here with a deviation therefrom. You will remember that I told you, when speaking of acute periositis, that there also the cartilage of the epiphysis, when it still exists, as is the case in young subjects, sometimes suppurates. If this occurs simultaneously at the upper and lower end, which it very rarely does, it is easy to understand that the sequestrum thereby becomes detached, and that it does so very early, so early that no formation of bone can have taken place around the suppurating cavity, or at least to a very slight extent only. If the bone is now removed, no equivalent has yet been nor will be furnished, because the required irritation is wanting, for it is the sequestrum which furnishes this irritation so long as it remains as a foreign body in the bone. Under such circumstances, therefore, the extremity may be deprived of bone and rendered useless if the sequestrum is removed very early. In cases of suppuration of the cartilage of the epyphysis at one end of the bone, *e.g.* at the lower end of the femur, the sequestrum continues attached above, and the softening of the bone must take place slowly here as in other places. If the sequestrum is not removed too early, the bony case formed around it may be very closely attached to the end of the epiphysis, and that so firmly that the lost piece of bone becomes thoroughly replaced thereby and its connection with the diaphysis quite firm and strong. We have observed this favorable result not long ago, in a youth of sixteen. It may happen, however, as I have once seen at a similar point of the thigh, that the lower end, detached at the cartilage of the epiphysis, presses from within upon the skin and gradually penetrates it so as to appear externally. The inferior epiphysis of the

FIG. 95.



Necrosis of the lower half of the diaphysis of the femur, with separation of the cartilage of the epiphysis and perforation of the skin.

femur was drawn upwards by the muscles and the appearance produced is represented in Fig. 95.

The sequestrum removed later on had the following form. (See Fig. 96).

FIG. 96.

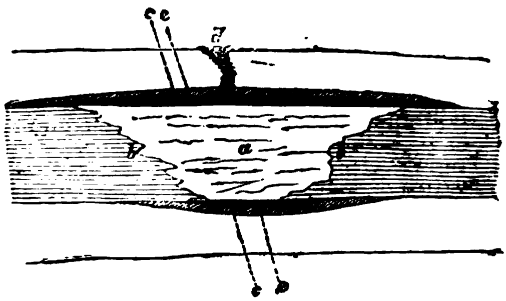
The sequestrum removed from Fig. 95.

The new formation of bone was strong enough to support the body later on, the knee was straightened under chloroform, and a complete cure was effected. I saw a perfectly similar case at the lower end of the humerus. In both cases the joint, *as is usual with necrosis in the vicinity of the joints*, had suffered severely, and became completely stiff. But also, although an especially early separation of the sequestrum may not occur in consequence of softening of the capsule of the epiphysis, the new formation of bone may, under circumstances of which we know but little, be very weak, so that, after the separation of the sequestrum, the new bone is not firm at one point, but quite pliable. There is then, therefore, a pseudarthrosis of the newly formed bone. I have seen two cases of this kind; one I cured completely by driving from time to time ivory wedges into the weak part of the newly-formed bone, which I thus stimulated to constant reproduction. This object was completely attained in the course of eight months, and the twelve-year-old boy afterwards walked as well as a healthy one.

I must point out clearly here that after osteomyelitis with necrosis in the vicinity of a joint (much more rarely after fractures in the same position), an excessive growth of the bones longitudinally has been observed, so that these bones become, in some cases, an inch longer than the normal bones of the other side. If the joint does not suffer much it sometimes becomes strikingly flabby and abnormally movable after osteo-myelitis, perhaps from an overgrowth of the ligaments of the joints. This state of things does not usually, however, interfere essentially with the use of the extremity, and comes to an end in the course of time.

More frequent than the necroses of the whole diaphysis just described are *partial necroses* of the same part, which may affect the whole thickness or only one half of the circumference, according to the extension of the osteomyelitic and periostitic process. You can easily apply what has been said already to these partial necroses. Here is an instance: periostitis in the femur has affected a portion of the diaphysis, and been followed by necrosis of the latter; the following state of things may exist (see Figs. 97 and 98):

FIG. 97.



Partial necrosis of a cylindrical bone. Diagrammatic drawing.

a, sequestrum; *b*, *b*, its boundaries; *c*, *c*, the suppurating cavity; *d*, the opening outwards; *e*, *e*, the thickened ossifying walls of the suppurating cavity.

Some months later (Fig. 98): *a*, detached sequestrum, which is to be removed; *e*, *e* newly formed bony mass to compensate for the piece which has been lost. The newly formed bone naturally covers the sequestrum anteriorly also, but had to be left out in the drawing, as in Figs. 90, 91, and 92, to show the sequestrum.

The process with which we have become acquainted here may also be taken as holding good for *necrosis in flat and spongy short bones*, but it must be remarked that, in the case of necrosis of these bones, the new formation is much less abundant, and is often wanting altogether. The inflammatory new growth in cases of disease of the spongy bones with necrosis generally very soon puts on an ulcerative character, and there is then no extensive new formation of bone. Very acute, non-traumatic periostitis is, moreover, very rare in spongy bones.

Also after periostitis and ostitis of a simply ossifying character originally, extensive necrosis may occur, namely, if the newly formed deposit of bone softens and becomes suppurative and

ichorous at the point where it is in connection with the diseased bone; the nutrition of the bone thereby gradually becomes greatly interfered with; it often lives on for some time in the neighbourhood of the medullary canal, or rather keeps up a sort of half

FIG. 98.

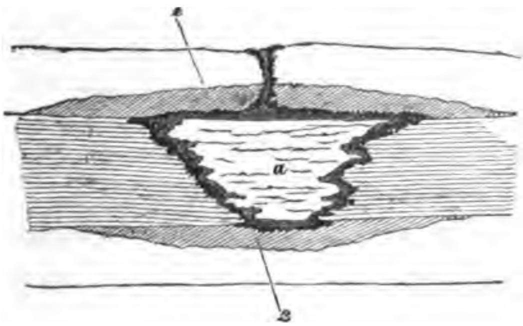


Fig. 97 at a later stage in the new formation of bone. Diagrammatic drawing.

existence between life and death. This form of periostitis and necrosis occurs especially in the maxillary bones after the chronic poisoning by the vapours of phosphorus, a disease peculiar to the workpeople in the manufactories of lucifer matches. I cannot go further here into the question of periostitis and necrosis resulting from phosphoric poisoning, which presents many remarkable peculiarities, because I should have to burden you with too many details which would confuse you now. If you bear clearly in mind at present the characteristics of necrosis in the cylindrical bones, you will have opportunities in the clinique of becoming familiar with the deviations which occur from special conditions in individual cases, since necrosis belongs to the comparatively most frequent diseases of the bones.

I cannot leave the subject of the anatomy of necrosis and the regenerations of bone which there take place without mentioning an excellent French surgeon who spent many years in studying the osteoplastic activity of the periosteum, and carried out very cleverly the earlier studies on this subject by Troja, Flourens, B. Heine, A. Wagner, and others: I mean Ollier, who has promoted these studies with indefatigable zeal experimentally and at the beside and exhausted them for a long time. I have repeated a part of his experiments, and can confirm from them the fact that, in young

animals, the conservation of the periosteum when bones are extir-

FIG. 99.

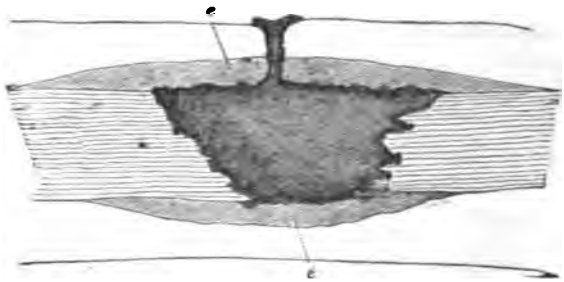
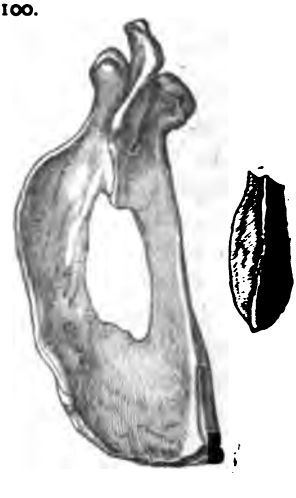


Fig. 98 after removal of the sequestrum.

pated essentially favours the regeneration of the bones under certain circumstances.

FIG. 100.



Scapula of a young dog, 150 days after removal of the piece designated, which, at the time of the resection, formed the whole of the bony portion of the scapula: the surface of the joint and edges of the cartilage, as well as the whole of the carefully detached periosteum, were preserved. Uninterrupted growth of the bone; almost complete regeneration of the resected portion.

Scapula of a young dog of the same litter, 150 days after the same operation, which was performed on the same day, but without detachment of the periosteum. Interrupted growth; no regeneration whatever of the resected portion.

As regards the osteoplastic power of the human periosteum, especially in comparison with other soft parts surrounding the bone, I have stated my opinion repeatedly in the course of these lectures, and find the views propounded on this subject confirmed by every fresh experience. New and interesting points of view concerning the growth of bone have quite recently been communicated by J. Wolff. He attempts, by variously modified experiments and reflections, to show that the growth of the bones is essentially interstitial, and is effected by means of a kind of slow expansion. His representation and conception of these conditions raised a lively discussion, and occasioned repeated examinations of the earlier diagrammatic conception of Flourens, according to which the growth of the cylindrical bones was brought about essentially by the cartilage of the epiphyses. The most recent experiments by Maas and Wegener have fully confirmed these older views.

We now pass on to the *symptoms* and *diagnosis* of necrosis. We call disease of bone necrosis from the moment when it is certain that a portion of a bone or an entire bone is dead up to the time of the removal of the sequestrum. The subsequent healing up of the bony cavity is for the most part a simple, healthy development of granulations with suppuration, which may, it is true, assume an ulcerative character. The question, therefore, is how we can recognise that a part is necrotic. This may, in many cases, be very easy, namely, when the necrosed bone is exposed to view; in all the cases, therefore, in which necrosis follows the laying bare of the bone. The dead bone appears perfectly white, but becomes in many cases also blackish, like other portions of tissue when drying up and becoming necrosed. Gangrene of bone may, as far as the bony substance is concerned, present itself in the dry form only; the soft parts in the bone, the vessels, the connective tissue, and medulla may, however, like other soft parts, be affected with either dry or moist gangrene; a complete desiccation occurs in almost all the cases in which the bone is freely exposed to the air. This necrosis superficialis is, therefore, seldom accompanied by decomposition or a bad smell. In more deep seated necrosis, *e.g.* of an entire diaphysis, or in sawn or fractured surfaces which are becoming necrotic and lie deeply beneath the soft parts, decomposition of the medulla generally ensues; the stench proceeding from a large

sequestrum on its removal is sometimes extremely powerful. The decomposing medulla is dangerous for the organism so long as no line of demarcation has been formed and the lymphatic vessels of the immediate neighbourhood are still pervious. If exuberant granulation occurs in the bone, at the boundary of the healthy parts, the layer of granulations forms a wall through which absorption does not easily take place so long as the granulation tissue continues healthy and does not itself undergo inflammation and gangrene. Now how do we recognise a deep-seated sequestrum? This can be done with certainty by means of a probe alone. We introduce through the openings at which the pus flows out as thick a probe as possible, and feel with it the surface of the sequestrum, which is generally smooth and firm, seldom rough or soft; we endeavour to push the probe along this surface to ascertain the length of the sequestrum. We next press the probe firmly against the sequestrum, to find out, if possible, whether it is detached and movable, or still quite firmly fixed; you will readily understand that this is important for determining whether we may already think of removing it. A further aid to diagnosis is the circumstance that the respective extremity is considerably thickened, and the massive new growth of bone can be felt; from the openings flows thick, yellow, often mucous pus; the bone is not particularly painful on pressure, nor does careful sounding usually cause pain, although the patient is often very much afraid of it, because many surgeons do it unnecessarily often, roughly, and yet without result. The patient is free from fever.

By these means you will, in many cases, easily succeed in diagnosing the necrosis; so long as no external openings exist, the diagnosis of central necrosis in a bone is always very uncertain. Necrosis can scarcely be confounded with anything except caries. The mode of origin and the locality do a good deal here to enable us to distinguish them, for necrosis occurs more frequently as a consequence of acute or subacute inflammation in cylindrical bones (femur, tibia, humerus), caries more often slowly in spongy bones or the spongy portions of bones. The objective symptoms also are different: in caries, there is little formation of bone in the circumference of the ulcer, often none that can be felt; in necrosis, there is considerable formation of bone; in caries, thin, unhealthy, serous pus is formed; in necrosis, mostly thick, often healthy, or mucous pus; in caries the probe passes into the friable bone, which is

generally rather painful; in necrosis the probe generally comes upon firm bone, and often causes no pain.

From this comparison of the phenomena which result from the different nature of the two diseases you must admit the possibility of a diagnosis, and in very many cases it is, in fact, extremely easy and simple. Other cases are more difficult to understand in reference to the anatomical conditions. If necrosis is combined with caries, all the phenomena rather point to caries, except that the necrosed portion may perhaps be recognised with the probe. In caries centralis of the cylindrical bones enormous thickening of the bone occurs in exceptional cases, and the inner wall of the bony cavity may feel very firm and hard, like a sequestrum. These cases may give rise to error: the cavity is opened, and no sequestrum is found, as had been expected. It is possible that in these certainly rare cases the sequestrum, which was perhaps not very large, had been absorbed, of which more presently. These exceptional cases, however, do not nullify the general rules, and you will do well for the present to rely upon the comparative diagnosis just propounded.

I will now say a few words concerning the fate of the sequestrum. What do you think? Might the piece of dead bone not be absorbed? Have I not told you repeatedly that dead bone might be dissolved, and carried away by the granulations? We might expect, then, that no assistance would be required for the elimination of the sequestrum. I have no doubt, judging from the observations I have made, that small sequestra may be removed completely by strongly developed granulations, but granulations in a constant state of decay or caseous degeneration have no power of dissolving and removing bone. We have already, when on the subject of caries, pointed out that partial necrosis so readily occurs in cases of atonic suppurative and cheesy ostitis because the inflammatory new growth, which decays immediately from deficient vascularity, does not dissolve the bone, which rather becomes, to a certain extent, macerated in the organism. The absorption of sequestra has its limits. First of all, naturally, no absorption takes place when the bone is fully laid bare, for the granulations do not act at all here; further, the absorption ceases as soon as the granulations begin to secrete pus on their surface. The sequestrum which is formed after acute periostitis will, therefore, at the point at which the periosteum suppurated away and pus is now secreted during the whole process, not generally be absorbed, because it does not come into close

contact with the granulations, but at all the points where the sequestrum becomes detached, an absorption takes place by means of the insterstitial granulation-mass which forms at the limit of the living portion of the bone. If this granulation-mass, when the sequestrum has become detached, begins to secrete pus, absorption ceases here also, and the sequestrum, which is now surrounded with pus, no longer decreases in size. The granulations in suppurating cavities which grow from all sides towards the sequestrum also become changed chemically in the course of time; they assume a gelatinous, mucous character, and very often undergo fatty degeneration. But the sequestrum must come out at last! Can it come out of itself? This happens sometimes; but what is the motive power which expels it? Imagine to yourselves a central necrosis, *e.g.* of the tibia; a sequestrum becomes detached on all sides, and is, then, for the reasons just mentioned, considerably smaller than the cavity in which it lies. The pieee of bone is now quite loose, granulations are growing towards it from all sides except from that on which the suppurating cavity opens outwards, there is no resistance there; if the opening be large enough and correspond to one end of the sequestrum the increasing granulations push the latter out at that point. For this, therefore, very special mechanical conditions are required, such as are seldom fulfilled. Small sequestra are not unfrequently expelled spontaneously; larger ones, which cannot pass through the existing openings, must be removed artificially.

The *treatment* of necrosis will consist at first simply in keeping the fistulæ clean. An artificial chemical solution of the sequestrum is not to be thought of. If you poured hydrochloric acid daily into the fistulous openings it would dissolve the newly formed bony substance from within, as much and indeed more than the sequestrum, which would be a great evil, since the new formation of bone must fill the place of the sequestrum. Nothing remains, therefore, but *the removal of the sequestrum mechanically. This must not be done until the sequestrum has become detached;* a very important maxim, which is based, firstly, upon the circumstance that the sawing off of the dead piece of bone is seldom possible without removing a good deal of the healthy and the newly formed bone, and, secondly, that the newly formed bone is seldom sufficiently firm before the sequestrum has become detached. The separation of the sequestrum does not take place generally until the new

formation of bone is sufficiently firm to take the place of the lost piece of bone. We must be careful not to endanger this important practical result of observation by a too active artificial interference. Only a few special exceptions to the above rule occur, chiefly in necrosis from phosphorus, but this is not a true necrosis, but very often combined with ichorous ostitis, of which I shall speak further when on the subject of special surgery and in the clinique. I have already told you that we can sometimes recognise with the probe whether a sequestrum is detached, but this is not always the case; the sequestrum may be so firmly compressed by the granulations as not to convey any feeling of movability; it is also difficult to verify movement in a very large sequestrum, and the curved shape of the bone, *e.g.* the lower jaw, may render this still more difficult. In such doubtful cases the length of the process and the thickness of the bony case aid greatly in distinguishing whether the sequestrum is detached or not. The majority of the sequestra generally become detached in from eight to ten months; in a year even an entire necrotic diaphysis usually lies loose as a sequestrum in the newly formed bony case. These are approximative leading rules which are naturally liable to exceptions. If the bony formation is still weak when the sequestrum is already detached, it is desirable in the case of the humerus, tibia, or femur, to defer the extraction, that the bony formation may gain strength, provided always that the general condition is not deteriorating. If albuminuria sets in, the extraction of the sequestrum should be hastened.

The extraction of sequestra, especially if it require preliminary enlargements of the cloacæ (the fistulæ which lead into the bony case), is called the *operation for necrosis* or *sequestrotomy*. This operation may be a very simple one. If one of the openings in the bony case is tolerably large and the sequestrum small, we take a pair of forceps with a firm grip, and, introducing it into the bony cavity, try to take hold of the sequestrum and draw it out; but if the openings are small and the sequestrum large, a portion of the bony case must be removed, both to admit of the introduction of instruments and of the withdrawal of the sequestrum. It is seldom possible, with the trephine, chisel, hammer, &c., to enlarge sufficiently a single opening. The following is the manner in which I generally perform the operation. With a short, strong resection-knife I make an incision through the soft parts down to the bony case, from one fistulous opening to another in its vicinity; I then

take an iron rasp with a handle and remove the thickened soft parts from the uneven surface of the bony case, so that the latter may be exposed to view to a certain length and breadth. This portion of the bony case is now to be removed to form an opening through which the sequestrum can be extracted. For this purpose saws of various kinds may be employed. The hammer and chisel have latterly always sufficed for me. Whatever instruments we may select, the work is always laborious. The portion of the bony case removed should be as small as possible, that its firmness may not be destroyed. When the bony case has been opened, we see the sequestrum before us and try to remove it with elevators or strong forceps—a work which is also sometimes very laborious. When this has been effected our problem is solved. If, contrary to expectation, the sequestrum is not detached, we must refrain from breaking its connections unnecessarily, and wait again some weeks or months until we have convinced ourselves that the exposed sequestrum has really become free. After the operation the suppurating cavity must be kept clean and the patient remain in bed for some time. The fistulæ now secrete much less freely than before, but it is often very long before the filling up of the cavity left by the sequestrum with ossifying granulations is completed. We cannot do much to promote this and the fistulæ, which remain long under such circumstances, usually cause so little trouble that we are not called upon to interfere actively with them. Sometimes, however, too large a hole remains open for a long time, its walls become sclerotic, and granulations cease to form. The treatment for atonic ulcers of bone is then indicated. The application of the ferrum candens to such old bony cavities, and the chiselling out of the bony fistulæ, are the only means from which I have now and then seen good results in such cases. Many such fistulæ in bone are incurable, but they cause no disturbances of function and had better be left to themselves.

The whole great importance of sequestronomy has only been fully appreciated during the course of the last decennia. It did not come into general use until after the introduction of chloroform, for the operation is a very violent one, and the chiselling, sawing, and hammering about the bony case appear horrible to a looker-on, especially as the operation may require a great deal of time. An amputation is a trifle in comparison with it. The local anæmia greatly facilitates the recognition of the anatomical conditions in

these operations. Formerly amputation was very often performed for total necrosis, even when no complication from joint affections existed—an idea which would probably not now occur to any surgeon. You therefore find in the older museums the most beautiful preparations of extensive necroses. These are now very rarely to be seen, because almost all sequestra are extracted at the proper time. The interference is a very important one locally, but the febrile reaction after it is generally very slight. Violent as the inflammatory symptoms and the fever would be if you acted in a similar manner with a healthy bone, this has very little effect upon the bony substance of the case of the sequestrum. I have seen only a single case in which evil results followed therefrom. I am convinced that the operation for necrosis is one of the most beneficial of operations, one by which many lives are saved which would have been lost after amputations, or from the general diseases which usually occur in the course of long-continued suppurations in the bones.

LECTURE XXXV.

Appendix to Chapter XVI.

Rachitis. Anatomical conditions. Symptoms. Ætiology. Treatment. Osteomalacia. Hypertrophy and Atrophy of the bones.

Rachitis and Osteomalacia.—We must now allude briefly to two other forms of constitutional disease which make themselves known chiefly by certain changes in the bones, namely, by softening and bending of them. These two diseases are called *rachitis* and *osteomalacia*. They are, in their effect upon the changes in the form of the bones, almost identical, but in their nature somewhat different. They are multiple forms of chronic inflammation, with peculiar characteristics.

Let us begin with rachitis, which term is taken from the Greek name for the spinal column, and signifies, strictly, inflammation of the latter. But the vertebral column seldom suffers much from rachitis; it is, therefore, by no means clear how the name arose. Rachitis was afterwards often called the "English Disease," because it was especially made known by English writers, and is also, perhaps, very common in England. The essential character of the disease consists therein *that the deposition of salts of lime in the growing bones is very deficient, and that the cartilages of the epiphyses are extremely thick.* You see from this already that the disease must be peculiar to early life; *it is a developmental disease of the bones,* but usually affects so many bones that it must be a question, not of local disturbances, but of a constitutional affection, which you may class with the dyscrasias already known to you. The insufficient deposition of salts of lime in the growing bones in rachitis is, however, also combined with an unusual development

of vessels, and especially with an unusual amount of absorption of the already formed bony tissue—a slight degree of absorption always occurs during the growth of the bones on the inner and outer surface of the cortical layer—and lastly also with unusually luxuriant growth of the cartilage of the epiphyses. If you add to this the fresh formations of osteophytes which are met with externally on the cylindrical bones, it cannot be denied that this disturbance of nutrition can scarcely be distinguished from the inflammatory form if a transition to suppuration and caseous degeneration also takes place.

In many cases we find rachitis in scrofulous children, and rachitis is, in fact, regarded by some physicians as one of the manifestations of scrofulosis. This is, however, not quite correct, for, on the one hand, we see in ricketty children none of the symptoms of scrofula, amongst which a disposition to swelling of the glands, suppuration, and caseous degeneration should be especially reckoned; and, on the other hand, the rachitic process has, anatomically, little relation to the forms of periostitis and ostitis such as we observe otherwise in scrofulous children, for rachitis never leads to caries. The disproportion between the growth of the bones and the impregnation of the bone tissue with salts of lime has, as a consequence, the want of sufficient firmness in the bones; they become bent, especially those which have to bear the weight of the body; with high degrees of softness of the bones the action of the muscles is sufficient to bend them. These curvatures occur most frequently in the lower extremities; the femur becomes bent convexly forwards and inwards, the bones of the leg convexly forwards, outwards, or inwards in their lower third.

The thorax becomes compressed laterally, so that the sternum projects sharply and the so-called "pigeon breast" is produced. Curvatures of the pelvis, the vertebral column, and even of the upper extremities also occur with high degrees of rachitis. The occiput of such children remains very long soft and impressible, and dentition is retarded. The softness of the occiput is, in many cases, the only symptom of rachitis, so that this affection has sometimes been regarded as entirely independent of general rachitic disease. Curvatures of the lower extremities depend, according to Virchow, chiefly upon a number of slight infractions of the whole bone, or of individual portions of the cortical layer. Complete fractures seldom occur; if they do, the cure generally takes place

quite firmly, under the usual mode of treatment, by means of bony callus.

FIG. 101.

Typical forms of rachitic curvatures of the legs.

Besides these curvatures in the bones, rachitis causes still other changes in them, namely, thickening of the epiphyses and of the points of union of the cartilages of the ribs with their bony portions. The thickening of the epiphyses may, for instance, be so great at the lower end of the radius that above the wrist-joint, corresponding to the point immediately behind the cartilage of the epiphysis of the radius, a second depression of the skin is formed. This appearance of the joints has given rise to the use of the expression "double joints." The nodular thickenings which occur at the anterior extremities of the bony ribs are very frequently visible, and since all lie regularly under each other, the name "rachitic rosary" has been applied to them. If these changes in the bones are present, we at once diagnose the existence of rachitis therefrom. Until one of the phenomena just mentioned is distinctly recognisable, the diagnosis is very uncertain. There are, it is true, certain prodromata: inordinate appetite, an enlarged abdomen, disinclination to

stand or walk, but these phenomena are too little marked to enable us to infer from them the existence of a general disease of the bones. The disease most frequently commences in the second year of life, and occurs in well-nourished and often even in fat children. Disturbances of digestion and a tendency to constipation are sometimes observable, but not always present. Of causal elements affecting the origin of rachitis we know very little; the disease occurs here in Germany pretty frequently in all classes of society, although more common amongst the poorer classes; in some cases it may be more or less hereditary; something faulty in the composition of the blood and in the assimilation of the food may be assumed hypothetically, but we have no proofs thereof.

As regards the course of the disease, I may remark that it often dies out under appropriate treatment, *i.e.* the curvatures of the bones do not increase, and the children, who had ceased to walk, again show an inclination to do so. In the further course of the normal growth of the bones the curvatures in them become always less and less marked, and slight degrees of curvature often disappear entirely; this is easy to understand from the mode of apposition-growth of the bones. Before the bones again attain their normal condition there generally occurs, towards the end of the rachitic process, an abnormally copious formation of bone, lasting for some time, so that the bones which had been rachitic are, at certain periods, unusually hard and firm and, as it were, sclerotic. In rare cases the rachitic condition continues until the skeleton is completely formed, and these are just the cases which occasion the extreme curvatures and displacements of the bones which are generally presented as types of this disease. In every pathologico-anatomical collection you will find examples of such entirely abnormal skeletons changed in appearance by rachitis. The greater my experience becomes, so much the more am I inclined to think that also the *flat-foot* formation, the development of the *genu valgum* and *varum*, and also many *lateral curvatures* of the vertebral column (skolioses) result essentially from a weakness of the bones which cannot be distinguished from a slight degree of rachitis. This variously localised rachitic condition occurs, indeed, in later years, *i.e.* chiefly in the second decennium of life, while the disease of the entire bony system briefly described as rachitis is met with, as already mentioned, mostly in very young children up to the sixth year. But it is a question, in both cases, of a continuing abnormal

softness, and of a certain yielding of *growing* bones which must, of course, be acted upon by various incidental causes for the production of such forms of curvature. You will often hear, later on, that many physicians place rachitis in direct relation to diseases of the brain, especially to paralysis, convulsions, and psychical derangements in children. I will not attempt to deny that this whole process of disease, as yet so obscure to us, may have a direct influence upon the development of the brain, but, in most cases, these relations are indirect. The rachitic process in the bones of the skull is often followed by a rapid tendency to sclerosis and such an extensive and active new formation of bone that even the sutures of many bones become ossified. The further uniform development of the skull thus becomes interrupted; the cranium becomes distorted and too small at certain points for the growing brain, and thus arise derangements of the function of the brain, because that organ is interfered with in its normal development by the rachitic cranium.

Rachitic children are seldom brought to the medical man until either the parents have been struck with the thick limbs or the curvatures, or until they, as the mothers frequently express themselves, "lose their legs," *i.e.* will no longer walk or stand, although they had already learnt to do so; the disease is so frequent and so well known that it is often recognised without asking the doctor. The *treatment* has generally only one object in view, viz. to correct the general unhealthy diathesis: it is, therefore, predominantly medical, and especially dietetic. As regards the latter point, the things to be avoided are: too free use of bread, potatoes, gruel, and flatulent vegetables. A good supply of milk, eggs, butcher's meat, good white bread, strengthening baths with malt, herbs, &c., is to be recommended. Cod-liver oil, steel, and similar strengthening and tonic remedies must be employed internally. Phosphate of lime has sometimes been recommended, sometimes declared to be useless. Beneke has found this preparation to be particularly beneficial for the cure of rachitis. I employ it on his recommendation, and give it mixed with an equal quantity of saccharine oxide of iron several times a day in milk or water. Children generally take it readily. From phosphorus alone also we might, according to the already mentioned experiments recorded by Wagner, have expected a cure for rachitis. It is always a rather one-sided view to assume, in forming an opinion concerning the rachitic and also

the osteomalacian process, that a deficient supply of lime alone is the cause of the non-deposition of lime in the growing bones, or of the disappearance of the lime already deposited. But it is also possible that the lime introduced into the stomach does not reach the blood at all on account of faulty processes of digestion, or that it becomes excreted by the kidneys to an excessive extent, or that the nature of rachitis consists therein that the newly forming bony tissue does not take up the lime furnished in normal or, perhaps, superabundant quantity. All these surmises do not, indeed, furnish any direct suggestions for treatment, but I mention them that you may see quite clearly that, in this case also, we are not justified, in a physiological point of view, in attributing the disturbance of nutrition exclusively to deficient supply.

Parents very often wish to have splints applied to remove the curvatures or, at least, to prevent them from increasing, and you will be asked whether the children should be made to go about or left in a recumbent position. As regards this point, it is best to let the children do as they like. So long as they do not wish to walk we had better not urge them to do so; if they lie down mostly, they must be kept as much as possible in the open air; to take a child out of a close dwelling in a town into the country for some time not unfrequently suffices to cure the rachitis. As regards the use of boots with splints and similar apparatuses which encumber the feet, they should be employed only in those cases of extreme curvature in which the position of the feet interferes mechanically with walking. This is rarely the case, and the use of such orthopædic contrivances consequently very slight in rachitis. If the rachitic condition has come to an end, such a great curvature may be left in very rare cases that it becomes necessary to do something to remedy it. In the majority of cases this is quite unnecessary, since the curvatures, as I have already informed you, disappear in the course of the further growth of the bones. In the case of the femur only, curvatures sometimes remain which cause such a displacement of the foot that only its inner or outer edge can reach the ground. If this state of things goes on for some years, we must attempt to remedy it. This may be done in two ways. We put the child under the influence of chloroform, and cautiously make an artificial, subcutaneous infraction of the bone; then, having the leg held straight, put on a plaster-of-Paris bandage, and treat the injury as a simple fracture; the healing process is

generally effected easily. In many cases, however, the bone has become so enormously firm after the cessation of the rachitis, that such an artificial fracture is not feasible. Subcutaneous osteotomy is then indicated. The results of this operation, which I have often been compelled to perform, have hitherto been extremely favorable. In most cases, the wound through the skin healed by the first intention and the treatment was then that of a simple fracture. The operation will, however, always be a rare one, because extreme curvatures from rachitis occur but seldom.

A few words more now on *osteomalacia,* or softening of the bones properly so called. This affection is also characterised by curva-

FIG. 102.

A woman suffering from extreme osteomalacia (after Morand). In the place of the bones were found mostly only membranous cylinders, or excessively thin bony tubes.

tures of the bones, but here a copious absorption of existing bony matter really takes place. The medulla goes on increasing in size, the cortical substance of the cylindrical bones becomes thinner and thinner, the bones consequently weaker and more pliable, and eventually a complete removal of the bone may occur, so that nothing but periosteum remains, which plays but a small part in the process, as only a small amount of osteophytes are formed by it. The spongy bones also become constantly weaker, the bony portion constantly thinner; they also become so soft that they shrink up on maceration. The medulla has a reddish, gelatinous appearance, but does not consist, as in fungous caries, of granulation-mass alone, but contains a great deal of medullary tissue. The appearances visible under the microscope during this process have already been described in connection with ostitis malacissans. Lactic acid has been found in the medulla of the cylindrical bones in osteomalacia, so that it is in the highest degree probable that the bone is dissolved thereby. The lime thus carried into the blood often passes off in large quantity with the urine in the form of oxalate of lime. You see from what I have just said that it is a question here of an ostitis malacissans, which presents nothing exceptional in its anatomical characters, but which owes its isolated position solely to the circumstance that it occurs in several bones at once under conditions which are quite peculiar, and that it never leads to suppuration or caseous degeneration.

As regards the ætiology of osteomalacia, very little is known about it. The disease occurs in certain parts of Europe, most frequently in women, in whom it becomes developed during pregnancy especially. Sometimes dragging pains and tenderness on every movement, or when the parts are touched, precede and accompany the disease in its further course. Curvatures occur primarily, and even exclusively, in the pelvis, which assumes in consequence a peculiar form of lateral compression, about which you will hear more in lectures on midwifery. Curvatures of the vertebral column and of the lower extremities, combined with contractions of muscles, supervene. The disease may present pauses, and recur during a fresh pregnancy, &c. Slight degrees and localised forms of osteomalacia, *e.g.* osteomalacia of the pelvis, not unfrequently heal spontaneously; if the disease be highly developed, general marasmus supervenes, of which the patient dies. The treatment is the same as that of rachitis, but the prospect of success is much slighter.

I will now allude briefly to *hypertrophy* and *atrophy* of the bones, which are more interesting anatomically than clinically.

Anatomically speaking, we may call every bone of which the longitudinal or transverse diameter is increased hypertrophic. It very rarely happens that individual cylindrical bones, *e.g.* a femur or a tibia, grow to excess longitudinally so as to produce inequality of the extremities; for this excessive growth the term "bony hypertrophy" is, to a certain extent, admissible; "giant growth" seems to me more applicable, but to use the term for every case of thickening or sclerosis, though it may be convenient anatomically, has no practical value, because these conditions of the bones may result from very varying processes of disease, which are partly still going on, partly completed. Almost more obscure is the notion of *atrophy* of bone; the term is sometimes applied, in an anatomical sense, to a carious, an osteomalaceous, or a half-destroyed bone, &c. This has no practical value. I do not mean to deny herewith that a wasting of the bones with true processes of inflammation may occur. Senile wasting of the bones, *e.g.* of the alveolar maxillary process, is a striking instance thereof. For this the term "atrophy of bone" may be retained; the expression may also be used when, in consequence of non-use of the limbs, *e.g.* in cases of paralysis, the bones become thinner and more porous (from containing more medulla), although this absorption is not combined with an increased vascularity and formation of osteophytes; their longitudinal growth is also considerably below the normal if the paralysis dates from very early youth. For all other cases it will be more correct to describe the process which causes the atrophy.

LECTURE XXXVI.

ON CHRONIC INFLAMMATION OF THE JOINTS.

CHAPTER XVII.

General remarks upon differences of the chief forms. A.—GRANULO-FUNGOUS *and* SUPPURATIVE *inflammations of the joints, tumor albus. Symptoms. Anatomy. Ostitis granulosa sicca. Ostitis with periarticular and periosteal abscesses. Atonic forms.—Ætiology.—Course and prognosis.*

IN chronic inflammations of the joints the synovial membrane is, no doubt, in half the number of cases, the part which first becomes diseased; in the other half the disease commences in the bone and in the ligaments of the joints. The affection of the synovial membrane may be accompanied by a greater or less secretion of fluid, and this fluid, again, may be purely serous, or rather of a suppurative character. Chronic dropsy of the joints is a form of disease which manifests itself chiefly by serous exudation, without any considerable destruction of the synovial membrane, and never passes into suppurative synovitis without some special external cause, any more than does chronic rheumatic inflammation of the joints, in which fibrous thickening of the ligaments and destructions and formations of cartilage and bone occur. But other forms of chronic inflammation of the joints may be combined from the first with suppuration, or are, if this is not the case, characterised by the formation of copious granulations with a tendency to pass into suppuration. The synovial membrane may then gradually become entirely converted into a mass of exuberant, spongy (fungous) granulations which, generally at least, secretes pus and causes purulent openings outwards—fistulæ, indolent abscesses—destroying the cartilage and bone and leading occasionally, therefore, to caries of the epiphyses. This latter group, which may also occur in somewhat different forms, we will call the *granulo-fungous* and *suppurative inflammations of the joint*; they are by far the most frequent

forms of all kinds of diseases of the joints, and will, therefore, occupy our attention for a considerable time. For a more minute detailed study of diseases of the joints generally, I would recommend to you especially the excellent works of Bonnet, Volkmann, and Hueter.

A.—*Granulo-fungous and suppurative inflammations of the joints. Tumor albus.*

Tumor albus, white swelling, is an old name which was employed formerly for almost every swelling of a joint which ran its course without reddening of the skin. At present this name, when used at all, is applied by general agreement for the forms of inflammation of the joints I am about to describe, which are further spoken of, especially in England, as *scrofulous* (strumous) *inflammations of the joints.*

The disease is very common in children, especially in the knee- and hip-joints; it commences mostly in a very lingering, more rarely in a subacute form. If, for instance, the knee-joint be affected, the first thing the parents usually notice is a slight dragging or limping in that leg; the child complains spontaneously, or when questioned concerning the limping, of pain after walking for some time, or on pressure upon the joint. A non-medical eye can detect nothing abnormal about the joint at first. The surgeon will observe pretty early, on comparing both knees, that the two furrows which are visible in the normal condition at the sides of the patella when the limb is extended, and give the healthy joint its beautiful form, are either effaced in the diseased knee or much shallower, at least, than in the healthy one. Beyond this there is nothing to be seen. The impediment to walking may be so slight that the children may go about for weeks or months limping a little, and make so little complaint, that the parents are not led until late to consult a surgeon; this is generally not done until the joint, after some unusual exertion, has become more painful and begun to swell. The swelling, which was scarcely observable at first, is now more easily recognised and the knee-joint is uniformly round and very painful on pressure. If we assume that no treatment is adopted at this period, but the disease left to run its course, the state of things will be about the following: the child drags itself about perhaps for some months longer, then a time comes at which it ceases to walk; it wants to lie down constantly because the joint is very painful; the latter

now gradually assumes more and more an angular position, especially after every subacute exacerbation. Single parts of the joints now become especially painful on the outer or inner side, or in the hollow of the knee; at one of these points distinct fluctuation presents itself, the skin there becomes red, and suppuration finally occurs from within outwards, which perforates the skin after some months; thin pus is discharged, mixed with fibrinous, cheesy flakes. The pain now becomes less severe, and the general condition improves; but this improvement does not last long—a fresh abscess is soon formed, and so things go on. In the mean time, from two to three years have perhaps passed, the general health has suffered considerably, and the child, which was formerly healthy and strong, is now pale and thin, and the successive abscesses are not unfrequently accompanied or followed by fever. With the development of each fresh abscess an exacerbation of the fever occurs, which weakens the patient more, the appetite decreases, digestion becomes sluggish, diarrhœa supervenes, and the emaciation increases from week to week.

The disease may even now, although rarely, spontaneously run a retrograde course; more frequently it progresses, and causes death from exhaustion, in consequence of the extensive suppuration and continuous hectic fever. If a recovery is about to take place, the following signs thereof present themselves: the suppuration decreases, the fistular openings contract, the general condition improves, the appetite returns, &c. Eventually the fistulæ heal, but the joint remains fixed at an angle or bent or distorted, but free from pain, and the patient escapes with life and a stiff joint. This termination of suppuration of a joint in *anchylosis* is the most favorable one possible in a severe case; the anchylosis itself may be complete or incomplete, *i.e.* the joint may be entirely immovable, or movable to a slight extent. The whole process will probably have occupied from two to four years. To the list of the local phenomena I must add that certain muscles in the neighbourhood of each joint gradually become permanently contracted; these are generally the flexors, in the hip-joint also, in some cases, the abductors and rotators, by means of which the patient places the joint in a permanent position which causes no pain or the smallest possible amount thereof. These pathological positions, which become developed to a greater or less extent according to the peculiarity of the individual case, may, if they result from muscular contraction alone and are not

of too long standing, be improved immediately under the influence of chloroform; but after months or years, shrinkings occur, first in the fasciæ, afterwards in the muscles, which can only be torn through under chloroform with the aid of considerable force. After long non-use of an extremity, the muscles eventually become highly atrophic from fatty degeneration and cicatricial shrinking. The capsule of the joint also, which had been much infiltrated and swollen, as well as the accessory ligaments, shrink up on that side of the joint especially towards which it was bent. In the knee-joint, therefore, this shrinking will be greatest in the hollow of the knee.

Comparatively rare are cases in which the disease commences with a sero-suppurative effusion into the joint (catarrh, blenorrhoic synovitis); I have seen this predominantly in tubercular subjects. The symptoms are then at first those of chronic dropsy of the joints, but the joint is painful and its function more impaired. Ostitis and periostitis in the neighbourhood of the joint not unfrequently gives rise to synovitis. One or the other side of the condyles of the femur or of the tibia, or of the lower end of the humerus, or the posterior surface of the olecranon becomes painful; the pain continues for a long time concentrated upon a certain point, then doughy œdema occurs, followed by an abscess. The joint sometimes remains for many months qnite intact in its function until the suppuration, sometimes accompanied by acute inflammatory symptomes, makes its way into the joint, and the same course of things as described above is observed. In many cases, these abscesses always remain periarticular and heal before perforation of the joint takes place. This often leads to periarticular cicatricial contractions, with thoroughly healthy joints.

Lastly, the bones also may become primarily diseased in the form of ostitis malacissans. This occurs especially in weakly individuals in the carpal and tarsal bones and in the head of the femur. The joints then frequently remain long intact, even when periosteal abscesses with great œdema and freely suppurating fistulæ become developed. In cases of primary disease of the external surface of the epiphyses, muscular contractions occur less frequently than in primary disease of the synovial membrane and in primary subchondral ostitis.

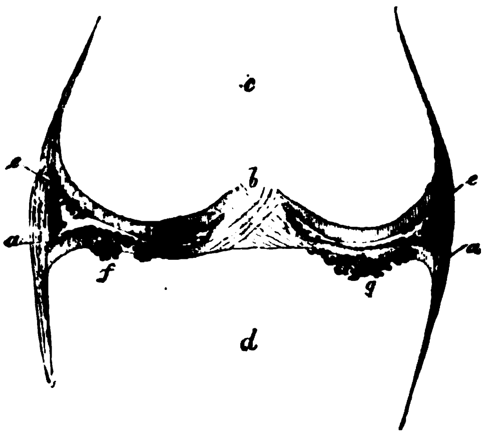
These brief sketches may serve as types to give you a preliminary notion of the disease in question and its importance. To enable you to understand the various forms in which it may present itself,

I consider it necessary, however, first to give you a clear idea of the anatomical course of things in these diseases of the joints. We have opportunities of observing this partly in resected joints, partly in amputated limbs, partly also in various stages in the post-mortem room. I have occupied myself so specially with this subject that I am in a position to give you an exact description of the anatomical changes from original investigations. These have many things in common in all cases, and from what you already know concerning chronic inflammation of other parts, you can easily imagine *à priori* that it will eventually prove to be a question again of a variation of the old story of serous and plastic infiltration, with varying degrees of vascularity, of exuberant growth, and of decay, &c.

Let us first examine these joints in various stages of disease with the naked eye. We will begin with the common case in which the process commences with chronic synovitis; we first observe swelling and redness of the synovial membrane; the latter is already changed in the lateral portions of the joint and in the folds of the membrane; its villi are thickened, but little lengthened, yet very soft and moist. The entire membrane is more easily distinguishable and separable than in the normal condition from the firm tissue of the capsule upon which it lies. The synovia during this stage is seldom increased in quantity, but turbid and somewhat similar to mucous pus. These changes in the synovial membrane gradually become more marked; it becomes thicker, more œdematous, softer, redder; the villi have grown into thick rolls, and already present here and there the appearance of spongy granulations. The cartilage loses the blueish lustre on its surface, but it is not yet visibly diseased. The synovial outgrowths now begin, however, to grow beyond the cartilage at the sides, and to insinuate themselves between the two opposed cartilaginous surfaces. In the meantime the capsule of the joint has also become thickened and assumed a uniform lardaceous appearance, and is also highly œdematous; this swelling and the œdema gradually extend also to the subcutaneous cellular tissue and the skin. Later on the changes in the cartilage occupy our attention chiefly; the exuberant growths of the synovial membrane gradually spread in the form of a reddish mass of granulations over the whole surface of the cartilage and cover it completely, lying upon it like a veil (Fig. 103); if we endeavour to remove this veil, we find it very firmly attached at some points by means of prolongations which these exuberant growths have pushed

into the cartilage, and which may best be compared to the roots which a shoot of ivy throws into the ground, or to the formation of

Fig. 103.



Diagrammatic section of a knee-joint (the interarticular cartilages are left out, the cartilages of the joint shaded) with granular inflammation in it; *a, a*, fibrous capsule; *b*, lig. cruciata; *c*, femur; *d*, tibia; *e, e*, fungous, exuberant synovial membrane growing into the cartilage, at *f* into the bone; at *g*, isolated exuberant granulation in the bone at the boundary between bone and cartilage.

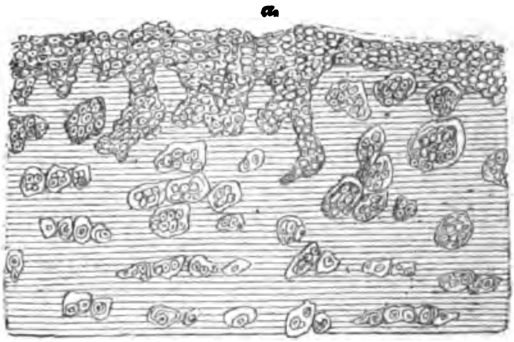
pannus upon the cornea (synovitis hyperplastica lævis seu pannosa, Hueter). These roots not only increase in length, however, but also spread out and gradually consume the cartilage, which appears, on the removal of the veil of exuberant fungous growth, at first rough here and there, then full of holes, but later on it disappears altogether, and the exuberant fungous growth then extends into the bone and begins to consume it; granular caries becomes developed in the form with which we have already become acquainted. The bone subsequently becomes absorbed, as already known to you, by the chronic-inflammatory new growth, and you thus have the transition and connection of granular inflammation of the joints with caries. The diseased process advances sometimes more at one point, sometimes at another; one condyle of a joint may be almost entirely consumed while another may still have retained a portion of its cartilaginous surface. As regards the remaining portions of the changed synovial membrane, they may also become developed to

excess outwards towards the capsule; the latter, the subcutaneous cellular tissue, and the skin become converted here or there into a mass of fungous granulations, and thus openings outwards and fistulæ are formed, which communicate either directly with the joint or with a synovial sac.

We will pause here a moment to consider what we observe with the microscope in the diseased parts; concerning this I can, at least, give you some fresh information. The normal synovial membrane consists of loose connective tissue, with a moderately extensive network of capillary vessels, which forms a complication of loops in the tufts; on the surface of the membrane is found a simple layer of endothelium with flat polygonal cells, as on most of the serous membranes. The tissue of the membrane gradually becomes occupied by cells, and at the same time softer; it also loses its tense fibrous character, and the vessels become dilated and considerably increased in number. The endothelium perishes as an isolated layer of flat cells; its place is taken by small, round, newly-formed cells, which soon blend with the tissue of the synovial membrane in a state of continuous transformation, and are then no longer distinguishable as a separate layer. The synovial membrane gradually loses entirely its former structure from the constantly progressing plastic infiltration; the connective tissue, occupied by countless new cells, gradually becomes homogeneous, and, from the steadily increasing vascularisation, the tissue now thoroughly resembles histologically that of the granulations. In these spongy granulations are formed here and there small white nodules, which behave partly like mucous tissue, and consist partly of pus cells and giant cells. In an anatomical sense there is no objection to calling these nodules "tubercles" (Köster), but we must at present hesitate to regard them already as the expression of that form of disease to which the name "tuberculosis" has hitherto been confined. Entirely similar processes go on on the surface of the cartilage, especially at those points at which it is covered with the granulo-fungous exuberant growth. The cartilage-cells are beginning to subdivide rapidly while the hyaline intercellular substance melts and becomes dissolved. If you cut from the surface of such a changed, perforated cartilage a piece horizontally, you will always find in the circumference of the defects a number of cartilage-cells in a state of exuberant growth, which is naturally combined with disappearance of the cartilaginous substance. At the points at which the cartilage has thus become

converted into a not yet vascularised cellular tissue, it blends with the exuberant synovial growth lying upon it; the latter sends off

FIG. 104.



Degeneration of the cartilaginous tissue in synovitis pannosa. *a*, granulation-tissue on the surface. Magnified 350 times; according to O. Weber.

vascular loops into it, and the more it thereby receives nourishment the more quickly does it consume the whole of the cartilaginous substance, and that in forms similar to those observed in lacunar corrosion of the bones.

You see from this description that the process of the destruction of the cartilage takes place in the same way as in bone, with the difference, however, that the cartilage-cells themselves take an active part in the destruction of the intercellular substance, while the bone-cells remain inactive, and the absorption is effected solely by

FIG. 105.

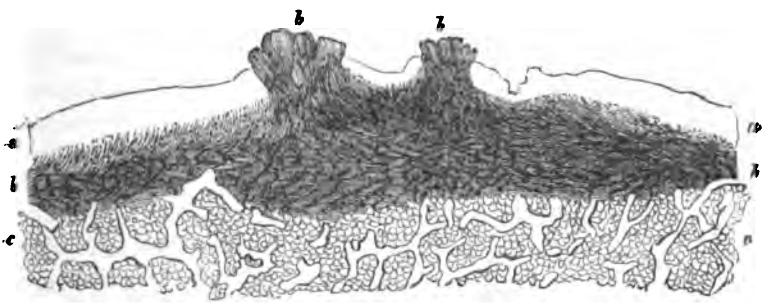
Atonic ulcerations of cartilage from the knee-joint of a child; the but slightly exuberant cartilage-cells undergo fatty degeneration and decay very rapidly together with the intercellular substance. Magnified 250 times.

the exuberance of the cells in the Haversian canals. But I must remark here already that forms are also sometimes observed in the

cartilage, from which it is evident that the cartilage-cells also sometimes take very little part in the exuberant cell growth, so that we must rather assume the existence of a passive absorption of the cartilaginous substance by the exuberant synovial growth. Whether, through multiplication of the cartilage-cells, movable pus-cells are also formed, is doubtful. In peracute ostitis and synovitis (panarthritis) the cartilage may also become necrotic and break up into plates and shreds before its cellular elements have reached the exuberant stage, as happens also in the cornea in peracute panophthalmia. As regards the histological changes in the capsule of the joint and in the accessory ligaments, these consist in serous and plastic infiltration, which only attains a high degree at a few points, but leads, for the most part, to a new formation of connective tissue, which presents to the naked eye the appearance of lardaceous thickening.

If the disease commences in the bone alone, or if the bone soon becomes implicated, it may happen that, simultaneously with the exuberant fungous growth in the synovial membrane, a similar growth becomes developed independently beneath the cartilage at the boundary line between it and the bone (Fig. 103), which communicates later on with that coming from above, so that the cartilage lies partly movable between the upper and lower layer of granulations. This is by no means uncommon, especially in the joints of

FIG. 106.



Subchondral granular ostitis of the talus. Breaking through of the exuberant granulations into the joint. Magnified 20 times. *a*, cartilage; *b*, masses of granulation; *c*, normal bone with medulla.

the hip, elbow, and foot; the cartilage becomes so completely separated by this *primary ostitis of the extremities of the bones,*

or *subchondral* caries, that it can be drawn off apparently tolerably intact from the often highly vascular soft bone beneath it. That inflammation of a joint may be caused by acute periostitis and osteomyelitis has been stated already; the inflammation then spreads from the periosteum to the capsule of the joint, and thence to the synovial membrane; the anatomical changes are the same as those described above. The infiltrations which we often meet with, for instance, on the back of the foot, about the sheaths of the tendons, and in the neighbourhood of the malleoli, are sometimes entirely independent affections of the periosteal and peritendinous cellular tissue, but their occurrence is often the result of ostitis of the carpal bones. Also, if an acute traumatic inflammation of a joint, or a spontaneous acute suppurative synovitis, passes into the chronic stage, the same anatomical changes occur as were described in connection with fungous inflammation of the joints. Traumatic periostitis in the neighbourhood of the joints may equally induce inflammation of the joints themselves if the suppuration-nests burst into them; the same may occur from chronic exuberant granulations in the capsule, *e.g.* the remains of ill-managed distortions of the joints.

Of great influence, *especially for the external form* of the diseased joints, is the question how far the parts in the immediate neighbourhood of the joint participate in the inflammation; if the capsule is much implicated, the joint will become uniformly swelled and round. This swelling is further increased considerably by the *formation of osteophytes,* which occupy the ends of the bones. These will be the more extensive the more the capsule of the joint and the periosteum of the ends of the bones are affected, and the more exuberant and productive the process is generally. While the condyles and the sockets of the joint are becoming destroyed from within, new bone forms externally, as you have already learnt in connection with chronic ostitis. But a considerable number of cases of caries of the articular extremities of the bones occur in which no osteophytes are formed. For caries of the joints another name is sometimes used which I have already mentioned to you, namely *arthrocacia;* this word is combined with the names of the different joints, and thus we have *gonarthrocacia, coxarthrocacia, omarthrocacia,* &c. Rust wrote a book on the diseases of the joints to which he gave the frightful name *Arthro-cacology,* of which you need not take any further notice. I mention it merely as a curiosity

which dates from a time at which also all knowledge of the diseases of the eye consisted chiefly in learning by heart the dreadful Greek names, a time which, fortunately, now lies far behind us. It is a point of great importance how much the muscles are implicated in a given case of tumor albus; in the neighbourhood of the inflamed joints, and often very extensively, the contractile substance in the primitive fibres wastes, generally after previous fatty degeneration, and the diseased limb becomes thinner and thinner, to a greater extent in some patients than in others. The more it wastes the more striking becomes the apparent enlargement of the joint, which is often found not to be very considerable on comparing it by measuring with the sound one. You will now and then hear and read of enlargements of the articular extremities of the bones in tumor albus, but the expression is an incorrect one; the bones never swell up in caries of the joints. If they appear to be thickened, this depends upon thickening of the soft parts or upon the deposition of layers of osteophytes.

A further difference in the course of the disease of the joint lies in the *greater or less tendency to suppuration*; abscesses and fistulæ *by no means necessarily* form a part of inflammation of the joints; they are always rather incidentals. You already know concerning ostitis granulosa, that it not unfrequently runs its course without suppuration. The granulo-fungous inflammation of the joints is pretty often combined with such an ostitis sicca; the process may go on for years, especially in otherwise healthy adults, without any formation of abscesses; extensive destruction of the cartilage and of the bones may take place, with the consequent displacements already spoken of in connection with caries, and yet not a drop of pus collect. If you examine the granular masses in the joint and in the bone in such a case, you will find them firmer than usual, sometimes of an almost cartilaginous consistence, similar to granulations about to shrink up and cicatrise and, in fact, a partial shrinking does occur in them, although the exuberant growth and, therewith, the destruction of the bone, often continue; such a process is related to cirrhosis. Suppuration is, therefore, by no means an absolutely certain measure of the spreading of the process in the bone; on the contrary, the more exuberant the growth of the granulations, the more extensive may be the destruction of the articular extremities of the bones. *The displacement of the bones*, the deformity of the joints, is the most important indication of the

extension of the process in the bone and in the ligaments. If, when the knee is diseased, the leg begins to become rotated outwards and the tibia to be displaced backwards, a large portion of the ligaments of the joint is generally softened or destroyed; if this abnormal position has existed for a long time, we may also infer with great probability that losses of bone have taken place. In very many, and, indeed, I may say in most cases, fungous inflammation of the joints is combined sooner or later with suppuration; the granulations either produce the pus within themselves, or it is secreted on the surface of a synovial sac not yet greatly diseased. Subacute synovitis sometimes occurs in individual sacs, whilst another part of the synovial membrane is still intact, a third already entirely degenerated. The knee- and elbow-joints are especially disposed to such defined, independent affections of individual synovial sacs, which communicate with the cavity of the joint by small openings only. Such suppurations are then for the most part combined with acute exacerbations of the pain and with febrile movements, especially if the abscess bursts outwards and synovial sacs previously but little involved in the inflammation become affected acutely or subacutely. *Early profuse suppuration in the joint* is, in many cases, a proof that the synovial membrane is, as yet, *but slightly* degenerated, for the greatest quantity of pus is secreted by serous membranes in the stage of suppurative catarrh. The pus secreted by the granulations of the synovial membrane is generally small in quantity and of a serous or mucous character. The state of things may be different if the suppuration, as is often the case, becomes settled in the cellular tissue around the joint and *periarticular abscesses*, which may, it is true, exist quite independently, without disease of the joint, supervene upon the fungous disease in the latter. All these suppurations acquire importance from the circumstance that they impair the general condition of the patient, partly by the discharges, partly by the fever.

In conclusion, we must now occupy ourselves briefly with the *state of vitality of the* inflammatory new growth and the anatomical consequences resulting from it. The vitality and luxuriance of growth and the further destinies of chronic inflammatory new formations depend, as you already know, very much upon the general constitutional circumstances of the individual, and that to such an extent that we can often draw conclusions from the condi-

tions of vitality in the local processes concerning the general state of health of the patient. Fungous inflammation of a joint, with caries sicca and a disposition to cicatricial shrinking of the new growth, will generally occur in otherwise healthy individuals, and in such cases we are often at a loss, especially when taking cold, over-exertion, or a mechanical injury of any kind is, perhaps, spoken of as the first cause, to explain to ourselves the chronicity of the process. The most luxuriant production of spongy granulations with secretion of mucous pus is also met with in tolerably healthy, or at least well-nourished individuals, and in stout, scrofulous children, also as the chronic continuation of an acute inflammation of the joints in persons who had been quite healthy up to that time, and only fell into an anæmic condition from the long continued suppuration. A strong tendency of the new growth to suppurative wasting or even to molecular decay is generally a sign of faulty nutrition; thin, stinking, profuse pus, with extensive, ulcerative destruction of the skin, and fistulous openings which look as if they had been cut out with a punch, is observed in inflammation of the joints, with or without caries, in old cachectic subjects, in ill-nourished tubercular subjects, and in atrophic, scrofulous children. The same thing may happen here as in indolent caries; the new growth is very short-lived; it is scarcely formed before it undergoes decay. Thus, together with the caries, necrotic processes are established, *e.g.* in the small carpal bones or, more rarely, in the epiphyses, accompanied, perhaps, by fatty degeneration of the new growth. We might separate this *atonic* form of chronic suppurative inflammation of the joints from the fungous form, but prefer not to do so, on the one hand, to avoid interfering with the general view, on the other, because this form also very frequently begins as extreme fungous synovitis and passes later on only, when deficient nutrition occurs, into the indolent form. We then find this form predominating on examination after death, and should overlook entirely the earlier condition if we did not take advantage of opportunities of studying it in resected and amputated limbs.

I will not enter further here into the anatomical details, which it would be easy to do, but what I have already said will suffice as a guide in each individual case. It is not impossible to group the various modifications of these processes to a certain extent in distinct classes, and to analyse them separately, but this does not

appear to me to be of any special importance, since these individual forms do not present distinctly either ætiological, prognostic, or therapeutic points for attack. In other words, if you have formed a correct notion of the anatomical course of things, and always recall to mind my description in every case which you have had an opportunity of seeing in the dead body, as well as in the living subject, in resected or in amputated limbs, &c., you will soon acquire a clear comprehension of this disease, and not require any further systematisation of the various forms in which it presents itself.

Of the *causes* of chronic fungous inflammation of the joints little more remains to be said beyond what you already know. The scrofulous diathesis very especially disposes to it; acute spontaneous or traumatic inflammations of the joints (whether the latter are caused by wounds, contusions, or distortion) sometimes pass into the chronic form. Scrofulous children from about the third year of life have a very special tendency to these affections of the joints; a fall, a drag upon the joint, or fatigue, not unfrequently furnishes the incidental cause for the breaking out of the disease. A whole series of cases remain in which we are unable to demonstrate any local or general causes. Thus I have met strikingly often in Switzerland with very atonic forms of fungo-purulent inflammations of the joints of the lower extremities in old people, without being able to to ascertain any cause thereof beyond, perhaps, the fatigue arising from climbing hills.

The *course* of the disease in question varies in a very extraordinary manner, but is always a chronic one lasting for *months*, generally for *several years*, combined frequently with pauses or retrograde formation, then again with exacerbations. At every stage of the disease, pauses or recovery may take place, and in its early stages, the latter may be complete, *i.e.* perfect movability of the joint may be restored (though this, unfortunately, is very rare), or the movability is partial only, *i.e.* a greater or less degree of stiffness remains. So long as the cartilage does not become too exuberant, or become destroyed from below by a new growth proceeding from the bone, the restoration of a tolerable amount of motion is possible, but this may, of course, be interfered with by cicatricial shrinking of the degenerated synovial membrane and of the infiltrated capsular ligaments, as well as by secondary contractions of the muscles. If the cartilage is partially or entirely

destroyed, and caries has set in gradually, or simultaneously with the commencement of the disease, recovery with anchylosis is alone possible, for cartilage does not form again here; the granulations of the opposed cartilaginous surfaces gradually blend with each other, and very tense adhesions are often formed, which may even become ossified. Whether things proceed so far, whether the destruction of the joint goes on continuously, depends very much upon the power of resistance of the individual patient; treatment may do much if commenced early, and the patient's general condition is not too unfavorable. The extent to which the muscles become implicated also varies greatly; the most powerful cause of atrophy of the muscles is met with, according to my experience, in those cases in which there is no suppuration of the joint but caries sicca occurs, and in which the affection of the joint proceeds from primary ostitis.

Let us now analyse briefly individual symptoms. Each form of this disease may run its course with more or less pain; upon what this depends I am unable to tell you; cases occur in which the bone becomes destroyed to a great extent without the existence of a trace of pain, others in which there is very severe pain; the more acute exacerbations with development of fresh abscesses are always rather painful. On examining the fistulæ with the probe, we sometimes come upon bone, sometimes not. Feeling the bone or not depends upon whether it is covered with granulations or lying quite free. As regards this point, I must refer you to what has already been said in connection with caries. The same holds good for the sensation of friction in diseased joints; crepitation is only of value as a sign of caries of the articular extremities of the bones if it is present; if absent, it furnishes no proof for the later stages that the bone is *not* diseased. The deformity, the displacement of the articular extremities of the bones, the *pathological or spontaneous luxations*, are the only tolerably certain indications of the amount of destruction of the bone. We can only be deceived here if the capsule has given way early and the head of the bone is really dislocated, a rare case, but one which has been observed in the hip, and may possibly occur in the shoulder also. For the formation of an opinion concerning the anatomical condition of the joint, we are almost reduced to what has already been said, but we help ourselves out with the aid of the ætiology, and especially of the duration of the whole process. Profuse suppuration from the joint itself is

always a sign that either a portion of the synovial membrane is not yet entirely degenerated, or that *large abscesses communicate with the joint;* the secretion from fungous granulations is less copious, and for the most part serous or mucous. For the degree of destruction of the cartilage we have no sure signs. To add anything special concerning the *diagnosis* of the disease and its *prognosis* would only lead to a repetition of what has been said already, in which you have all the means for forming an opinion completely at hand. My experience has taught me further that a slight degree of swelling of the joint, with great pain and early atrophy of the muscles in anæmic children, but little or no suppuration, indicates primary disease of the bone and renders the prognosis most unfavorable. A good state of nutrition is the chief ground for a favorable prognosis, which is not always greatly interfered with by early and even extensive suppuration

LECTURE XXXVII.

Treatment of tumor albus. Operative measures. Resections of the joints. Critical appreciation of these operations in the different joints.

Let us now direct our attention to *treatment*. This must, as in all chronic inflammations, be both local and general, and the latter must be the more relied upon when the constitutional disturbance is most marked. Concerning this general treatment itself nothing more need be said here, as the main points in it are already known to you. The state of nutrition of the patient, anæmia, the general hygienic and dietetic conditions in which he lives, must be the chief points of attack for the treatment. It is your duty to give the best advice you can to the patient in this respect, but you will soon learn that it is just in these points that you will meet with the greatest indifference, and that your advice about them will seldom be followed. We are especially helpless in regard to the worst influences, namely, hereditary tendencies, for we cannot possibly expect that a time should come in which only the strongest individuals of healthy families shall be chosen to propagate the species, and all weakly members of unhealthy families forbidden to marry.

As regards the local treatment and its results, it may be remarked that it is in general the more efficient the more acutely the disease run its course; it is for the most part not difficult to overcome subacute exacerbations or subacute beginnings of the process. The remedies mentioned so often already act extremely well here: strong ointments of nitrate of silver (5 parts to 40), painting with tincture of iodine, flying blisters, ice, hydropathic wrappings, slight compression with bandages. To this must be added absolute rest for the joint, which can only be attained for the lower extremities by constantly keeping the patient quiet in bed. I have no personal

experience as yet concerning the applicability of shampooing at the commencement of tumor albus, but it might be useful if employed cautiously. I cannot help fearing, however, that if practised a little too violently in such cases it might promote suppuration where there is a tendency thereto, and would therefore advise you provisorially not to adopt this mode of treatment except in the more indolent cases. If the process does not improve after a certain period of rest and the employment of the above-named remedies, I know of no better means than *the application of continuous moderate pressure upon the swollen limb by means of a firm bandage, generally a plaster-of-Paris bandage, which at the same time keeps the joint perfectly still in a proper position.* We may allow patients to go about with such a bandage if they have no pain; a stick or crutch, according to the amount of weakness which the patient feels in the diseased leg, serves as a support. If it is thought desirable to use baths at this period, the dressing must be cut open longitudinally, taken off before the bath, and reapplied after it. If the pecuniary means of the patient admit of it and there is an intelligent and skilful bandagist at hand, the dressing may be replaced by various kinds of light splint apparatuses, which not only keep the diseased joint at rest, but must be so constructed as to relieve it as far as possible of the weight of the body. Mechanical skill has made very satisfactory progress in this direction also. With these auxiliary means we may allow patients with disease of the lower extremities to take some exercise daily. This has the advantage that the muscles of the limb are brought somewhat, at least, into motion, and do not, therefore, become so much atrophied. We must not believe that in consequence of wearing plaster-of-Paris dressings or splints for a considerable time stiffness of the joint must always necessarily result; the contrary is by no means uncommon, namely, that a limb which admitted of very little motion before the dressing was applied is more movable after its removal than before. The reason of this is that the swelling of the synovial membrane sometimes undergoes a retrograde process beneath the dressing. Before the dressing is applied the limb should be well rubbed with mercurial or nitrate of silver ointment, or a mercurial plaster put on. I cannot too strongly recommend the plaster-of-Paris dressings in fungous inflammations of the joints for all the cases running a very chronic course; this treatment appears very insignificant, but is highly efficient in comparison with all the other means at our dis-

posal for combating this disease. I can assure you that since I have carried it out consistently I have met with fewer cases of suppuration and formation of fistulæ. Even if there is distinct fluctuation, you must still apply the dressing; you will seldom find, indeed, that these abscesses become absorbed, but if they burst spontaneously under the dressing, which the patient easily recognises by the soaking of the latter, this occurs more easily and unobservedly, without any increase of the disease or pain, than with any other kind of treatment. If fistulæ form, the dressing must still be employed, but it must be cut open and lined afresh with wadding; it must be removed daily, that the wound may be cleansed, and then reapplied; the general strengthening dietetic treatment must, at the same time, be carried out consistently. If the limb is very painful and fistulæ exist, perforated dressings should be used. I have by this means sometimes saved limbs with a fair amount of motion and favorable position which presented at first the most unfavorable prognosis, and have indeed frequently myself been very agreeably surprised at the results of this mode of treatment. The extension of suppurating or otherwise greatly diseased joints must always be done with great caution, and if we meet with resistance even under the influence of chloroform, should never be carried out completely on one occasion, but only so far as is feasible without strong pressure upon the articular extremities of the bones. In cases of disease of the knee and hip, I employ, with excellent results, the so greatly recommended slow extension by means of weights, and sometimes thereby prepare the patients, especially if children, for the application of the dressing. Volkmann has recently gained great credit in reference to the treatment of the diseases of the joints by his energetic recommendation of this mode of treatment, to which he has given the name of *distraction-method*. He attaches especial importance to the circumstance that the extension lessens as much as possible the pressure of the surfaces of the joint upon each other produced by muscular contraction and shrinking of the ligaments. The manner in which the extension is produoed is of such great importance for the practical applicability of this method that I must advise you strongly to pay especial attention to the mechanism employed for that purpose in the clinique. There can be no doubt that this method is more efficacious in most cases of commencing and progressing disease of the joints than that with plaster-of-Paris dressings, and you will, therefore, see it employed

especially often in my clinique; but, on the one hand, you will not always persuade patients in private practice to adopt at once the recumbent position, and, on the other hand, the method itself requires so much careful watching on the part of the surgeon that its applicability is, unfortunately, somewhat lessened thereby. A very ingenious American surgeon, Taylor, has constructed machines for the lower extremities, by means of which the distraction is effected and the joint unburdened, while the patient is still able to go about. These apparatuses frequently answer extremely well, but they are difficult to make, and the employment of them requires a certain amount of experience on the part of the surgeon. All the mechanical aids just mentioned—plaster-of-Paris dressings, apparatuses for support, distraction-dressings, Taylor's machines—require constant superintendence on the part of the surgeon to prevent the formation of wounds by friction or pressure, or evil consequences from displacement of the apparatuses. It requires inexhaustible patience and perseverance to ascertain, in children, whether the extension is sufficient or too powerful, to accustom them to the inconvenience of the apparatuses, to pacify the over-anxious parents if the child cries from ill-humour or weariness, to obtain obedience at one time by friendly persuasion, at another by a very earnest manner, and to prevent them from displacing the apparatus, &c. It very seldom happens that this mode of treatment is carried out consistently in private practice, and we cannot, therefore, too strongly advise its being done in hospitals or orthopædic institutions, at least until the chief dangers of curvature are obviated. Perseverance on your part and on that of your patients is absolutely necessary for the cure of chronic inflammations of the joints. Represent from the first to your patients that it is a question of a process which will go on *at least for several months, perhaps for some years,* and that the treatment may only cease when the limb is quite free from pain and strong enough for use, whether with or without motion. As regards indolent abscesses, I advise you once more *only to open them* if you intend to operate afterwards; if this is impossible, or it is not your intention to do so, wait for their spontaneous bursting, even if years should be required therefor.

If I have hitherto communicated to you briefly only my own maxims for the treatment of fungous inflammation of the joints, I must not omit to call your attention to the fact that other surgeons follow different therapeutic rules. We still meet with adherents to

the strictly dogmatic, antiphlogistic treatment, surgeons who, even in chronic inflammations of the joints, occasionally apply leeches or cupping-glasses, or employ cold applications and purgatives. They afterwards pass on to cataplasms, and end with moxas and the ferrum candens. If the disease still advances, and fistulæ have formed here and there, and the patient has become very anæmic, the indication for amputation is at once complete, especially if crepitation can be detected in the joint. This was the earlier standpoint, the results were favorable or unfavorable as we choose to express it; the former in so far as the amputations which, under such circumstances, were generally made pretty early, usually turned out well. I am inclined to attribute the more favorable results and less frequent indication for amputation to a more careful consideration of the mechanical conditions in the treatment of diseases of the joints, and feel certain that a great number of limbs are thereby saved in a comparatively useful state which would certainly have been amputated formerly. As regards local abstractions of blood in chronic diseases of the joints, I can by no means recommend them; they may be of some use in subacute exacerbations only, but we possess much better remedies in such cases, which have no ill effects resulting from them, for it is certainly detrimental to take blood, and that frequently perhaps, from subjects whom disease already disposes to anæmia. Cold is, under certain circumstances, of great advantage in subacute attacks of chronic inflammations of the joints; I employ, by Esmarch's advice, ice with good results in such cases, but it is difficult to keep a patient for years in bed in the same position with a bladder of ice on his knee, especially if he has little pain in the limb. I must also speak of the employment of a *continuous high temperature* as attained by carefully applied cataplasms, warm water dressings, cataplasms of hot peat (*e.g.* in Franzensbad), or mud (in Ofen, Pystian). This mode of treatment may be indicated if the course of the process is an extremely indolent one, or if a moderate degree of stimulation appears desirable for ill-looking fistulous cavernous ulcers, or deficient vascularity in the granulations with unhealthy thin secretion. In any case, the higher degrees of temperature, when employed at all, must not be kept up too long, because they otherwise lose their effect and, instead of the increased action you wish to produce, complete relaxation sets in.

You may conclude from the effects of treatment just described

that the results in cases of fungous inflammation of the joints are, in general, tolerably favorable if we leave out of consideration the greater or less degrees of stiffness left behind, and, above all, *if the patient comes early under treatment.* There remain, however, a long series of cases which, in spite of the most careful treatment, either do not heal at all, or, after an improvement of short duration, undergo exacerbations, and some even which, after completely healing, suffer relapses; the causes of this lie partly in the anatomical condition of the diseased joint, partly in the general condition of the patient. Diseases of the joints of the hand and foot are, for anatomical reasons, the most unfavorable; on account of the many small bones and joints which are here involved, the process is generally extremely tedious; the disease commences perhaps in a very chronic form in one of the carpal or tarsal joints, remains for a long time stationary there, or even undergoes partially a retrograde formation. A fresh joint now, perhaps, becomes affected; suppuration sets in here and there; the patient becomes anæmic, weak, condemned to inaction for years, and finally himself wishes earnestly to have amputation performed that he may again feel comparatively well after such long suffering. In other cases, a cachectic condition soon sets in which runs its course with anæmia and great disturbance of digestion, and ends with lardaceous disease of the internal organs or tuberculosis of the lungs, &c., so that, with such general constitutional conditions, a cure cannot be expected. If, under such circumstances, we quietly let the disease go on, the patient succumbs sooner or later, and more quickly in proportion as the affected joint is larger (the knee or hip), or a greater number of joints are affected at the same time, which is not unfrequently the case. There are two ways of still affording help under such circumstances: (1) to abandon the joint for the purpose of saving life, *i.e.* to amputate the limb; (2) to abandon the hope of healing the joint, to cut out the diseased ends of the bones for the purpose of saving both life and the limb, *i.e.* to perform resection of the diseased joint.

If we compare these two means *à priori* with each other, there can be no doubt that we shall prefer resection to amputation, and, in principle, this is quite correct; modern surgery is justly proud of the development of resections of the joints. Many unfavorable circumstances may, however, exist which nevertheless induce us to give the preference in a given case to amputation; the chief of

these is the extent to which the general health of the patient has suffered. After resection of a joint we have a large wound with two sawn surfaces remaining, which, in any case, will suppurate for weeks, and sometimes for months. Suppuration of the subcutaneous cellular tissue and sheaths of the tendons, suppurative periostitis and necrosis, or even caries of the sawn surfaces may supervene, all things which the patient may perhaps overcome, but which at all event require time and strength. If, therefore, the loss of power in weakly, cachectic individuals indicates the necessity for operative interference, amputation is often a more certain means of saving life than resection. *The preservation of life must always rank higher with the surgeon than that of the limbs.* We have, therefore, to decide whether the patient can bear resection and its consequences well. The answer to this question is difficult to give in a general way, and may be difficult even in an individual case; we possess no means of ascertaining the power of resisting disease. We have to examine whether the patient has simply become greatly emaciated, anæmic, and weak from the drain upon his system, or whether more deeply seated affections of internal organs exist. In the latter case amputation will be preferable if it is not too late even for this, for it is self-evident that we must not operate upon atrophic children with disease in several joints, indolent abscesses, diarrhœa, aphthæ, &c., or in individuals with tubercular cavities in the lungs, in patients with indurated, lardaceous liver or spleen, any more than in old, altogether wasted (marantisch) people. We must not deceive ourselves in such cases concerning the impotency of our art. We have further to consider what operation will least endanger life. This is a question which does not admit of a general answer. We must take into considerstion the particular joints which we propose to resect, and compare the operation with the amputation which might become necessary in the case in question. In caries of the *shoulder-joint* resection is less dangerous than exarticulation of the arm at the shoulder; the same holds good for the *hip-joint.* Exarticulation of the leg at the hip is one of the most dangerous operations; resection of the head of the femur in young subjects is not very dangerous. In the case of the shoulder and hip, therefore, exarticulation on account of caries is quite out of the question; what we have to consider here is whether the general condition of the patient is such that we may let the disease run its course, or whether we should cut short the process by means of resection. In the most

favorable case of spontaneous healing, anchylosis in a bad position will follow; if the cure takes place after resection, the extremity generally remains movable at the shoulder or hip, and the limb is, in favorable cases, tolerably useful. These chances speak strongly in favour of resection, especially in the shoulder-joint; we might here even decide pretty early in favour of resection for the purpose of curing the patient quickly and well. As regards the hip, there is one serious objection to the resection of this joint; we cannot remove at all or to a very insufficient extent only the acetabulum, which is generally also affected. The resection, when the joint is greatly diseased, remains incomplete; slighter degrees of disease heal without operation.

Much more favorable, perhaps most so of all, are the experiences recorded for the *elbow-joint*. The resection of this joint is not more dangerous than amputation of the upper arm; after resection we generally have, in favorable cases, a tolerably useful joint, after spontaneous healing, almost always anchylosis. Here the choice is easier; we shall more readily decide in favour of resection of the elbow-joint, not because the operation was necessary on account of serious danger to life, for caries of this joint only threatens to prove fatal when the disease *goes on very long*, but because it offers the chances of a movable, useful joint in a shorter time, with comparatively little danger to life, while the expectant treatment generally ends, after some years, in anchylosis. Some have even gone so far as to saw out anchylosed joints to form movable false ones. I should not recommend this, for the experiences concerning the usefulness of arms with resected joints have shown that the false joints formed after the operation often become looser and looser in the course of years, so that the limb operated upon eventually remains less useful than we had previously assumed it would be. Unfortunately, we cannot feel certain of the final result in reference to the usefulness of a resected extremity, although we can do much for its improvement by means of artificial supports, gymnastic exercises, and electricity. Very different is the state of things as regards the *knee-joint;* the resection of that joint is a more dangerous operation than the resections of joints hitherto mentioned, and about equals, in that respect, the deep amputations of the thigh. After resection of the knee-joint, all we hope to attain is anchylosis, which also results from spontaneous healing of the joint. This operation, therefore, since it does not effect more, with a considerable amount of danger,

than what may also be effected by non-operative surgical treatment if the process comes to a standstill, must only be undertaken when necessary for the saving of life; hitherto I have but seldom determined upon an operation on account of caries of the knee-joint, whether amputation or resection; only when every kind of treatment has remained useless for years, and the patient has become emaciated and suffers greatly, can there be any question of amputation, or in the case of old people in whom the cure of highly developed caries of the knee-joint is improbable. A resection can only be performed with success in young subjects with a good constitution; it is generally only a means of accelerating the healing process in cases in which the chances are comparatively good.

These are my personal views, which become constantly more confirmed the greater the number of cases of disease of the knee-joint which I see heal spontaneously. I have already seen many children die from coxitis, and am, therefore, inclined to recommend resection of the hip-joint, although the results of my own operations for it have not hitherto been favorable; after caries of the knee-joint I have seen old and wasted people and individuals with tubercles in the lungs and large cavities die, but more rarely children. Other surgeons hold very different views on this subject, and in England especially resection of the knee-joint is thought so highly of that the operation is performed very frequently there, and in early stages of the disease. Many German surgeons will, I believe, agree with me on this point; others occupy a middle position, and judge more favorably of resections of the knee-joint after a few such operations have proved successful. I was formerly directly opposed to this operation, but have changed my opinion somewhat after a series of favorable results obtained by me with it during the last few years. If we select for operation cases which offer favorable chances, and do not operate in doubtful or unfavorable ones, we shall not operate often, but generally with success, and shall of course cure but few by this means. An entirely analogous state of things exists in reference to many of the great operations; if we have some experience, and don't mind sending out most of our cases uncured, and if we interest ourselves especially for the more favorable cases, we shall soon get the reputation of very successful operators and surgeons.

We now come to the *wrist-joint*; the resection of this joint will

consist, in most cases, in the extirpation of all the carpal bones and the sawing off of the lower articular surface of the radius, perhaps also of the articular surfaces of the metacarpal bones. I have performed this operation several times, partly with brilliant success; the hand became perfectly movable again, the fingers useful. Two of the female patients were sempstresses, and both of them followed their occupation as before. A third and fourth patient unfortunately lost patience; when, after resection, the wound had closed, with the exception of two fistulæ, and the pain had ceased, they withdrew themselves from further treatment; some carious points still remained in the metacarpal bones which ought to have been extirpated, after which the result would doubtless have been as good as in the other cases. I should have been very glad to perform resection of the hand more frequently, but have several times been defeated by the determined will of the patient to have the forearm amputated. It must appear strange that a patient should not willingly consent if the surgeon proposes to him, by means of a tolerably safe operation, such as resection of the wrist-joint is, to save the hand. I was always obliged, it is true, to remark that several months would elapse before the hand healed, that the patients might not expect more than our art can do, and received the answer that they could not wait so long; they had already been without the use of the hand for four, five, to eight years, and always suffered pain; they were tired of treatment, and had decided upon having the hand removed, for which reason they would not enter anew upon a long course of treatment. I have told you this that you may see what difficulties present themselves to the surgeon, however honestly he may strive to do the best for his patients. By no means are all cases of caries of the wrist-joint fitted for resection; before any considerable destruction of the bones has taken place we should not decide upon an operation, even although we can predict that caries of the wrist-joint especially seldom heals spontaneously with movability. Caries of that joint is altogether infrequent in comparison with gonarthrocacia and coxarthrocacia, and occurs especially seldom in children, but more frequently in adults. The reason why healing occurs with so much difficulty lies partly in the local circumstances, as has been mentioned already. Moreover, there are so many tendons about the hand, the sheaths of which almost all become involved, and that often to a great extent; the fingers remain stiffly extended, and the metacarpal bones and

the radius and ulna are often affected, though perhaps only periostitis exists in them. The softs parts about the wrist are generally perforated by a great number of fistulæ, and even destroyed to a considerable extent, so that the favorable conditions for resection are thereby lost. *In cases of very extensive caries of the hand, with considerable degeneration of the surrounding soft parts, therefore, amputation of the forearm will reassume its ancient position.* The extraction of individual carpal bones, or the sawing off of the end of the radius alone, seldom serves our purpose. I have seen cases, it is true, in which the disease was confined to one or two of the carpal bones; these had become necrotic, and the process had stopped there. I extracted the bones, and healing took place very quickly in one case; the patient had been sent to me to have the hand amputated, and was very glad when I was able to tell him after the first examination that amputation was not to be thought of. These cases are rare, however; in general the diseased process goes on and cannot be checked by the removal of individual bones more seriously affected. On the whole I am of opinion that the total resection of the wrist-joint is not performed often enough, and that it is an operation which, according to my observations, deserves the greatest attention of surgeons. We may correctly apply to this operation, as well as to similar operations in the foot, of which we shall have to speak presently, an argument which has incorrectly been applied to resections in general, viz. if the resection does not bring about the termination of the local process of disease, amputation always remains as a last refuge. This holds good for resections of the hand or foot, in which pyæmia is seldom to be feared, but not for the shoulder, hip, elbow, or knee; if these operations are not successful, and suppuration becomes exhausting or pyæmia supervenes, little is to be hoped for more from amputations or exarticulations.

We now come, lastly, to the *ankle-joint*, and understand thereby all the joints of the tarsus, as well as the tibio-tarsal joint. The state of things closely resembles that observed in the wrist-joint. Although caries of individual bones of the carpus, *e.g.* the not uncommon caries necrotica of the calcaneus, heals spontaneously in the course of time almost with certainty, especially in children, as does also scrofulous caries of the fingers, toes, metatarsal and metacarpal bones, caries of the joints of the foot and of the larger tarsal bones seldom heals spontaneously, even in young grown-up

people, and scarcely ever in old subjects. Operative interference will, therefore, frequently be indicated here sooner or later, and a superficial consideration might lead to the belief that there would be a wide field for resections and extirpations of bones. Experience, however, has furnished two reasons against too wide an extension of such operations for caries of the foot, namely (1), the observation that after the extirpation of one bone the disease very frequently passes to another, and thus no complete cure is effected; (2) the circumstance that the foot must always retain so much firmness that the patient can walk upon it. We may, therefore, extirpate the ossa cuneiformia, the os naviculare or os cuboideum, or even the talus *or* the calcaneus, but to remove both talus and calcaneus, and perhaps also saw off the articular surface of the tibia would, even if healing took place, leave a rather useless foot, which is of less value than a good amputation-stump. The cicatrices which take the places of the extirpated bones shrink up in time very much, and if a certain amount of bone is formed in this cicatrix, no such regeneration occurs as after necrosis, but the foot contracts greatly at the point where the bone is wanting, and is thereby bent and rendered useless. These are, therefore, serious obstacles, in addition to which a good stump, such as remains, for instance, after Syme's or Pirogoff's method of exarticulation, is often quite as good or safer perhaps for walking than a weak bent foot, while the latter generally requires several months, the former six to eight weeks. In one case I removed all three ossa cuneiformia and the os cuboideum with very good success, and in other cases have extirpated the talus in boys; the tibia there articulated with the calcaneus, the new joint remained movable, and the patients did not even limp in walking. Such results speak strongly in favour of these operations. On another occasion I wished to remove the calcaneus alone on account of caries, but found, contrary to expectation, the talus also much diseased from below, and was thus obliged to remove the latter also. The result was most unsatisfactory; the youth lay for six months in the ward and there was no sign of healing. I then took off the leg low down, and healing took place by the first intention. Some weeks later the patient went out cured, with a good wooden leg, very glad to have got rid of his diseased foot. Above all, the extremely favorable results of Pirogoff's method of amputating compete strongly with resections of the ankle-joint, and I believe that experience will soon decide more generally than at present

against the too great extension of extirpation of the tarsal bones, and in favour of amputation in cases of disease about the foot.

Resections of the joints, which have come so much into favour during the last thirty years, had at first something so dazzling about them on account of the favorable results in individual joints, especially in those of the elbow and shoulder, that the performance of them may here and there have been carried to excess; this is the fate of everything which the human intellect invents; now only are we arriving gradually at more certain indications for these operations. It was, of course, necessary first to collect data, and it soon became evident that the resection of each individual joint possesses a very different value. Although I will by no means assert that these data are already to be regarded as sufficient, I believe that I have given you in what I have said above a correct summary of the present state of things. The general tendency now is less to attempt the formation of movable false joints after resections than formerly, but more frequently to be content with bringing about anchylosis of the joints, as quickly and with as little suppuration as possible, by partial removal of the portions of bone chiefly affected with caries by Lister's method of treatment.

There is one remark which I cannot refrain from making here at the end of this chapter. Since the patients in whom resection or amputation had been performed successfully on account of caries in the Canton of Zurich have come under my observation frequently later on, I have seen with pain that many of them who, after years of suffering, had left the hospital thoroughly cured and in good health, returned after one or two years with caries in other bones, or tubercles in the lungs, often not to leave the hospital again. The final terminations of diseases of the bones and joints are, unfortunately, much less favorable than they are generally assumed to be. Relapses, even in joints which have been cured with anchylosis for years, are unfortunately also by no means uncommon. Individuals who have suffered from these forms of chronic inflammation of the joints seldom live to an advanced age; you will meet few persons over forty or fifty years of age with anchylosis after scrofulous tumor albus. I see just in this circumstance further

evidence that these diseases are connected with constitutional states of the body, however difficult it may be to show this in all cases and to demonstrate it to those who are inclined to regard all diatheses and dyscrasias as the useless invention of the older theorists.

LECTURE XXXVIII.

B. *Chronic serous synovitis. Hydrops articulorum chronicus. Anatomy. Symptoms. Treatment. Typical recurring hydrops genu.* APPENDIX. *On chronic dropsies of the sheaths of the tendons, of synovial herniæ of the joints, and of the subcutaneous mucous pouches.*

B. *On chronic serous synovitis. Hydrops articulorum chronicus. Hydrarthron.*

THE chronic diseases of the joints of which we have still to speak are all much more rare than the already described granulo-fungous synovitis with its consequences and combinations, ostitis and caries of the articular extremities of the bones. The following diseases are, all taken together, scarcely so frequent as the former ones, and are in so far to be put in contrast with the fungo-suppurative inflammations of the joints as a connected group, as they never lead spontaneously to suppuration except when repeated irritations or injuries act upon them. Tedious and distressing as they often are for the patient, they have yet no connection with the most serious constitutional affections, such as tuberculosis or lardaceous disease, and therefore seldom prove fatal. They are, moreovcr, less diseases of youth than of middle age.

We will begin with the simplest of these forms, *chronic serous synovitis* or *hydrops chronicus articulorum*, or *hydrarthron*. The disease consists in an abnormal, rather slowly increasing collection of somewhat thin synovia; the synovial membrane becomes very little changed, it gradually becomes somewhat thicker, denser, and the connective tissue increases, but without much vascularity; the entire pathological changes in the tissues are extremely slight, even when the disease goes on long. Many surgeons refuse altogether to classify these dropsies of the joints, as well as similar affections

of the mucous pouches, with the chronic inflammations, but place them, as peculiar diseases, amongst the anomalies of secretion. This does not appear to me well founded. No one can doubt that chronic catarrhs of the mucous membranes, with predominant hyper-secretion, are to be classed with the chronic inflammations; chronic dropsy of the synovial membranes is completely analogous to chronic catarrh of the mucous membranes.

As regards the origin of chronic dropsy of the joints, it is very frequently a relic of an acute hydrops articuli after contusion, exposure to cold, &c., as has been mentioned already, or sometimes occurs in joints which had previously been diseased chronically and cured; in many cases, however, the disease commences at once in a very chronic form and remains chronic. Whether gonorrhœa has any connection with this disease I leave an open question; the cases of inflammation of the knee-joint occurring with gonorrhœa which I have seen were rather of a subacute kind.

Hydrarthron occurs spontaneously chiefly in young men, by far most frequently in the knee, and often on both sides; it is very rare in the shoulder, hip, or elbow; I have never seen it in a genuine form in other joints. If the disease is highly developed it is easy to recognise; the joint is very much swollen, with fluctuation at all points; in the knee we have also the flapping of the patella, which is raised up by the fluid, and may easily be pressed against the fossa intercondylica, sometimes with an audible splashing sound. Since the surfaces of the joint are connected with each other by firm ligaments (in the knee by the lig. lateralia and cruciata), which do not easily stretch, the fluid collects especially in the mucous pouches of the joint, and thus the nature of the swelling may often be diagnosed by the sight as dropsical, especially in the knee, where the bursæ under the tendon of the extensors on both sides of the patella and in the popliteal space are greatly distended by the fluid, while, on the contrary, in the case of uniform swelling of the capsule, there is a more regular appearance of roundness. Moreover, patients with this kind of dropsy can move the joint pretty freely and without pain, or even make long marches with it, and sometimes have so little inconvenience therefrom that they do not ask for any advice at all. Examination of the joint by palpation also gives no pain. After unusual exertion, a feeling of fatigue in the limb is experienced in advanced dropsy of the joint, as well as some pain and increase of exudation; this goes off again, however,

after a little rest, and the inconvenience is thus generally very slight.

The *prognosis* is always in so far a favorable one as these dropsies of the joints lead to nothing more; the fluid may increase enormously, but there the matter ends, and if no over-exertions or injuries supervene, things remain as they are. As regards the curability of the affection, the prognosis in this respect is most favorable for all those cases in which the disease remained behind after a subacute or acute commencement; in these cases a complete cure by means of absorption generally takes place, although slowly. Very obstinate, on the contrary, are the cases in which the disease commences and runs its course quite chronically; they are often very difficult to cure, sometimes quite incurable.

The *treatment* consists in the employment of the remedies already known to you in connection with chronic inflammations, &c., which must be used persistently while the joint is kept thoroughly at rest. These are, tincture of iodine, flying blisters, hydropathic wrappings of the joint, compression. The latter is the most efficient remedy, but it must be powerful and carried out consistently. The joint must be firmly covered with wet or elastic bandages; the vessels in the hollow of the knee are to be defended from pressure by a splint slightly bent and hollowed; the patient must remain in the recumbent position during the treatment of several weeks' duration; if moderate œdema of the leg sets in it is of no consequence, but if the toes become blue and cold, the bandages must be removed. If the patients will not submit to such a course of treatment, from which we can by no means absolutely promise a cure, we put a mercurial plaster around the knee, over which we let them wear a leather knee-cap, which prevents too violent motion of the joint and gives firmness to the limb in walking. If all this has done no good after being employed for months or years, or if the results of the treatment have always been of short duration, we still have simple puncture left us, or puncture followed by compression, or by the injection of iodine. Simple puncture generally does little good; you take a fine trocar and push it into the joint near the patella, then let the fluid flow out slowly, but close the canula before it has all come away, to prevent the entrance of air into the joint. You now close the wound with plaster and paint the joint immediately with tincture of iodine, after which you put on tightly wet bandages, or a collodion dressing.

You may thus effect a cure in individual cases; a rapid collection of serum, combined with some pain, will take place in the joint, and this fresh fluid may then gradually become completely absorbed.

If this operation has been of no use, and the fluid collects again and remains unchanged, you can then try puncturing, followed by the injection of iodine. This operation is, indeed, not entirely free from danger. It is performed in the following manner:—The puncture is first made carefully as described above, then a well-made syringe is filled with a mixture of equal parts of the officinal tincture of iodine and distilled water, or, if you wish to be particularly cautious, with one part of the tincture to two of water. Of this mixture you inject, after having thoroughly convinced yourselves that there is no air in the syringe, from 40 to 80 grammes, according to the extent to which the joint was distended, allow it to remain from three to five minutes according to the amount of pain in the joint, and then let it flow out again; then follows the exact closing of the wound and the bandaging and fixing of the limb. A fresh, acute, serous exudation now sets in and continues for about a week at the same point, after which it gradually becomes absorbed and a complete cure is in most cases effected. That the patient during such a course of treatment, just as after simple puncture, must remain absolutely at rest is a matter of course, for inflammation always sets in, and in all inflammations of the joints rest is the first condition for a cure. How it happens that tincture of iodine, although it may remain only a short time in contact with a serous membrane disposed to excessive secretion, acts in such an alterative and repressive manner upon the further secretion, is by no means clear. It was formerly believed that, after these injections, which were employed with advantage in many chronic dropsies of serous membranes, an adhesive inflammation and actual adhesion of the serous surfaces took place and produced complete obliteration of the sac. This is by no means the case, at least after the successful injection of iodine in hydrops articuli; if such an adhesion occurred the joint would become stiff. The process is a different one: the iodine is precipitated upon the surface of the membrane and in the endothelial cells, remains there sometimes for months, and appears to check further secretion by its presence. At first a strong fluxion with serous exudation takes place (an acute, serous synovitis), but the serum becomes absorbed by the still distended

vessels and the membrane shrinks later on from thickening of the connective tissue to its normal volume. A thickening of the synovial membrane always remains, however. This is about the way in which we must represent to ourselves the healing process, from analogy of the same process as it frequently occurs in the tunica vaginalis propria testis and produces hydrocele of the tunica vaginalis. After injections of iodine in hydrocele, frequent opportunities have occurred for investigation from which the mode of healing appears to be such as has just been described above. The shrinking of the serous membrane, with new formation of the endothelium, appears to me lastly to be the reason why the secretion does not continue. The *injection of iodine* in hydrarthron is not often practised by many surgeons; I have seen it done three times, and done it twice myself, and always with a favorable result, but this is not always the case. There are a number of cases on record in which the operation had no effect, and had to be repeated. I must warn you not to let these repetitions follow too closely upon each other, but to let the acute stage after the previous operation pass off. Other cases are known in which, after these injections of iodine, which were especially often practised in France, where they had been introduced by Boinet and Velpeau, *very violent inflammations of the joints* occurred; the acute serous synovitis became converted, as is so often the case with traumatic inflammation of the joints, into an acute suppurative one, and in favorable cases healing with anchylosis followed; *in some cases amputation became necessary, in others the patients died of pyæmia*. The unfavorable results after an operation which is undertaken for a disease obstinate, it is true, but by no means dangerous to life, have justly deterred surgeons from injecting iodine into the joints, and *I am, therefore, far removed from recommending this operation strongly to you*; it always remains connected with danger to the joint and to life, and should not, therefore, be performed without a very clear indication.

The *diagnosis* of hydrarthron is, in most cases, simple, and the disease differs greatly, as already stated, from chronic, fungo-purulent synovitis. I must point out to you, however, that at the commencement of tumor albus, serous exudations to a moderate extent and even fluctuation in the joint occur, so that the differential diagnosis cannot at first be formed exactly; an observation of some weeks suffices, however, to form a clear idea of the nature of the disease, which is further facilitated by the circumstance that hydrops

articulorum predominates in young grown-up subjects, while tumor albus is especially frequent in children.

A very rare and strange disease is the typical, relapsing hydrops genu, which has been described repeatedly. I saw it recently for the first time in a young man who had dropsy of the knee every nine days, with subacute symptoms; the fluid became completely reabsorbed in five days with simple rest, and the joint then remained entirely normal for four days; this had been thus repeated for nearly four months when I saw him; he had had gonorrhœa not long before the commencement of the disease. I had no opportunity of observing the case further.

APPENDIX.

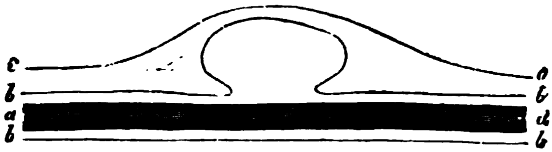
On chronic dropsies of the sheaths of the tendons and of the subcutaneous mucous pouches and on synovial herniæ.

We will now *speak of chronic dropsies of the sheaths of the tendons.* The disease consists therein that the synovia, which is secreted by the sheaths of the tendons to keep the motion of the tendons free and smooth, collects in an abnormally large quantity and greatly distends the sacs of these sheaths. Such a dropsy affects most frequently the sheaths of the tendons of the flexor muscles of the hand. A swelling gradually forms, partly in the palm of the hand, partly at the lower end of the anterior surface of the forearm, and we can feel quite distinctly how a fluid in the sheaths of the tendons, from the palm of the hand to the forearm, can be pressed backwards and forwards beneath the lig. carpi volare. The fingers are generally in a flexed position, and cannot be fully extended; the movements of the hand and fingers are somewhat less powerful than usual; there is no pain, and the patients do not actually consult a surgeon until the disease had become highly developed.

Another form of this disease *is the partial, hernia-like distension of the sheaths of the tendons with dropsy.* There forms in the sheath of one of the tendons a sac-like projection, sometimes as large as a pigeon's egg, with an abnormal collection of synovia.

This is called in common surgical language a *ganglion*. It is a much more common disease than dropsy of the whole sheaths of the tendons, but its occurrence is confined to some particular spots.

FIG. 107.



Diagrammatic drawing of the most frequent form of ganglion. *a*. Tendon. *b*. Sheath of tendon with hernia-like projection upwards. *c*. Skin.

The ganglia are most frequently situated upon the dorsal surface of the wrist-joint and proceed from the sheaths of the tendons of the extensor muscles; they are rare on the palmar surface of the hand or higher up on the forearm; and lastly, much more rare on the foot, where they are comparatively most frequently met with on the sheaths of the tendons of the peronei muscles. The contents of such a ganglion consist, in most cases, of a thick, clear, vitreous-looking gelatinous mass. The ganglion does not always originate in dilatation of the sheaths of the tendons, but it appears that, in other cases, the gelatinous mass forms, in a manner not yet understood, in the immediate neighbourhood of the sheaths of the tendons, and then first enters into communication with them. The contents of the above described larger distensions of the sheaths of the tendons may also consist of perfectly clear gelatine, but it often happens that a great number of white bodies resembling grains of rice or melon seeds are observed, which are entirely without organisation, and generally consist of purely amorphous fibrous tissue. These bodies may be present in such an enormous quantity that little or no fluid passes out when a puncture is made into the sacs. In many cases we can diagnose the presence of these grains of fibrine with certainty *à priori*, since they produce, as in subacute inflammation of the sheaths of the tendons, a very distinct friction sound.

In the *treatment*, we must especially bear in mind that under no circumstances should any operative interference be undertaken which might produce suppurative inflammation of the sheaths of the tendons, by which the patient, hitherto but little inconvenienced by the tumour, might be thrown on to a sick bed for a long time,

and possibly be left with a perfectly stiff hand. The remedies which are capable of so strongly promoting absorption in acute and subacute inflammations, such as mercury, tincture of iodine, &c., have scarcely any effect here. The simplest, and therefore, most frequently adopted operative procedure is the *crushing of the tumour.* If the ganglion, as is usually the case, is situated on the dorsal surface of the hand, we take hold of the flexed hand of the patient, place both thumbs close together upon the ganglion, and apply strong pressure, by which means the sac of the ganglion is sometimes burst and the contents poured out into the subcutaneous cellular tissue, where it readily becomes absorbed. Against this method there is little to be objected for those cases in which it succeeds readily, except that the evil is not always radically cured. The small subcutaneous opening soon closes again of itself, the fluid collects afresh, and the evil exists again in the same form as before. If we do not succeed in bursting the sac of the ganglion in the way above described, it has been suggested that the bursting should be effected by means of a hard blow with a broad hammer. In those cases in which the sac is too thick to be burst, I adopt the method of *subcutaneous puncture;* I take a very thin, short, curved, pointed knife (Dieffenbach's tenotome), push it horizontally into the sac, and scratch the inner wall of the latter repeatedly with the point of the knife; I then withdraw the knife slowly and at the same time press the fluid out of the sac. After this, I at once apply a compress, bandage the hand and forearm with a wet roller to prevent much movement, and let the patient carry the arm in a sling for four or five days. The bandage is then removed, the small punctured wound has healed, and the ganglion does not generally form again, as usually happens after simply emptying the ganglion. The extirpation of the whole sac by an external incision has been performed frequently, sometimes successfully without any considerable consequent inflammation. In some cases, however, suppuration of the respective tendinous sheath has occurred, or loss of motion of the fingers has followed, so that I do not recommend this method. The differences in the results after extirpations of these sacs may depend upon there being a free, or a very slight, or *no communication whatever with the sheath of the tendon;* that the latter is sometimes the case I have become convinced by occasional investigations after death.

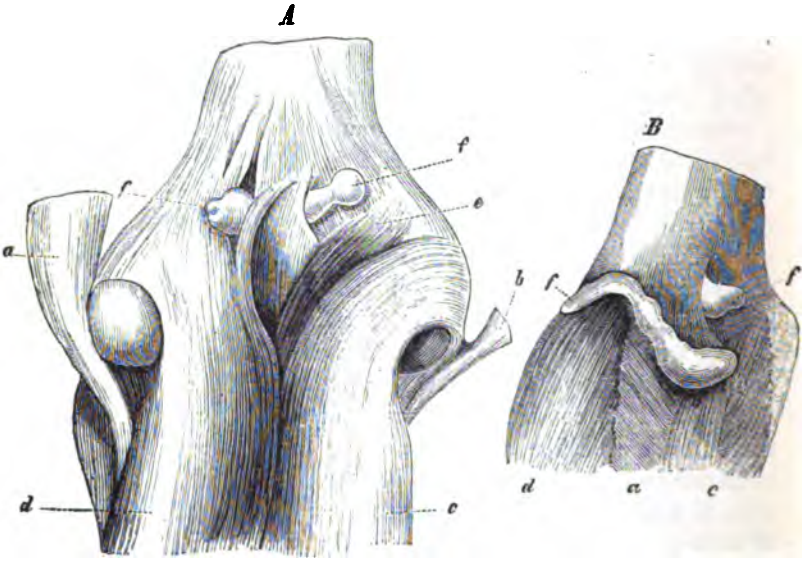
The treatment of extensive dropsies of the sheaths of the tendons

in the palm of the hand and forearm is infinitely more difficult; since the subcutaneous puncture is not advisable for several reasons, and the employment of absorbents has very little effect, nothing remains but to have recourse to methods which, in many cases, may cause suppuration, though to a slight extent only. Ask yourselves beforehand, therefore, whether there is any necessity for interfering. If the disturbance of function is not so considerable that the patient is thereby interrupted essentially in his occupation, you had better not interfere. If you are compelled to do something, however, your choice lies almost necessarily between two things, namely, between a free incision and a puncture with consequent injection of a solution of iodine. If you decide upon making a puncture, which I recommend in preference to an incision, you must use a middle-sized trocar, because the fibrin corpuscles will not pass out through a very fine one. You will often find it difficult to get them away even then, and will render the process more easy by injecting from time to time a little tepid water through the canula into the sac. The quantity of the fluid discharged is, as already stated, often very considerable; on one occasion I have withdrawn a tumbler and a half full of fluid from the sac in the sheath of a tendon. When the fluid has all been withdrawn, the syringe is to be filled with half an ounce of tincture of iodine mixed with an equal quantity of water, or with a similar quantity of a solution of iodine and iodide of potassium. This is to be injected slowly, allowed to remain from one to two minutes in the sac, and then allowed to run off again. The canula must then be taken out, the wound covered with a small compress, the hand and arm carefully bandaged and then fixed upon a splint. The patient must remain several days in bed. A considerable amount of swelling will again take place from a collection of fluid resulting from acute inflammation of the serous sac. If the tension be very considerable, the bandage must be removed, the punctured wound carefully closed with a piece of plaster, and the swollen parts painted with strong tincture of iodine. In the most favorable case, the tumour will then gradually diminish in size and become less painful and entirely disappear in the course of a fortnight or three weeks. In many other cases, however, suppuration, though transient, will set in, which may be successfully checked and finally stopped by means of ice. In the most unfavorable circumstances, however, extensive, deep suppuration of the sheaths of the tendons may occur, with necrosis of the

tendons and its consequences. A free incision through the whole tumour has frequently been made in recent times and with good results: moderate suppuration, healing with a tolerable amount of motion in the fingers. Extensive, progressive suppuration of the sheaths may, indeed, also occur, with unfavorable consequences for the function of the hand, or even with a fatal termination from pyæmia.

I must mention here that *hernia-like bulgings* may occur in the *capsules of the joints* also, quite similar to those in the sheaths of the tendons, which may become dropsical in themselves, while the dropsy does not extend to the whole synovial membrane. The fibres of the capsule become detached from each other, and through this slit the synovial membrane passes in the form of a finger of a glove into the subcutaneous cellular tissue. Although such forma-

FIG. 108.



Hernial bulgings of the synovial membrane of the knee-joint backwards (according to W. Gruber). *A a.* M. semimembranosus. *c d.* M. gastrocnemius. *e.* M. plantaris. *f f.* Hernia of the synovial membrane. *B b.* Capsule of the knee-joint. *c d.* M. gastrocnemius. *f f.* Hernia of the synovial membrane.

tions may occur occasionally in various shapes in connection with all the joints, they are generally met with especially in the knee-,

wrist-, and elbow-joints; I have frequently observed in the latter these isolated dropsies of hernial sacs of the synovial membrane communicating with the joint. Slight stiffness of the joint and a moderate amount of arthritis deformans were connected therewith.

I strongly advise you against any operative interference with these ganglia of the joints; such an operation may be followed by suppuration of the joint.

Cartilaginous bodies, chondromata, partly in a state of ossification, occur in villi of the sacs of the sheaths of the tendons; a formation of lipoma (lipoma arborescens, J. Müller) has also been observed in the villi. These new formations should only be extirpated when the inconvenience they occasion is considerable.

We will speak here at once of chronic dropsies of the *subcutaneous mucous pouches*. On opening such a bursa by means of a wound through the skin, suppuration from the sac frequently sets in, which goes on for a considerable time, but is seldom followed by any dangerous consequences, although suppuration may extend thence into the subcutaneous cellular tissue and become troublesome by its long continuance. When the greater part of the wound in the skin has healed, a small opening remains through which a probe can be passed into the sac; from this *bursal fistula* a moderate quantity of serum flows daily. The healing of these fistulæ may sometimes be effected by cauterisation with nitrate of silver, and compression by means of adhesive plaster. In many cases, however, they obstinately resist treatment; you may then attempt, by the injection of tincture of iodine, to effect a somewhat more intense suppuration of the internal surface of the sac, and obliteration of the latter from shrinking or adhesion; a shorter method is, however, to introduce a probe-pointed knife through the fistula into the sac, and to lay open entirely it and the skin above it, so that the whole of its inner surface may become exposed. Granulations will then gradually spring from it, the sac will shrink, and the wound will eventually cicatrise. I very decidedly prefer this shorter mode of proceeding, which is, at the same time, free from danger.

Quite analogous to the dropsy of the sheaths of the tendons just described is *dropsy of the subcutaneous bursæ*. Pressure or blows now and then give rise to it; in many cases, however, it is not possible to ascertain any cause for it. Although dropsy may occur in all the normal as well as in accidentally formed subcutaneous

mucous pouches, it is especially common in the bursa præpatellaris, which consists, according to the investigations of Linhart, in many cases, of two or three such pouches lying one above the other, partly entirely separate, sometimes communicating with each other. Dropsy of the bursa præpatellaris is very easily recognised, because the tumour, which attains to about the size of a small apple, is situated very distinctly upon the patella, and it is easy to ascertain that the sac in which the fluid is contained does not communicate with the knee-joint. This disease frequently commences as an acute or sub-acute inflammation: the fluid collects rapidly, the tumour is painful, the skin over it somewhat reddened, and the patient much inconvenienced in walking. The terminations may differ greatly; complete absorption frequently takes place, and things return to their normal state. In other cases partial absorption occurs; the phenomena of acute inflammation disappear, and the condition gradually becomes chronic. One of the rarest terminations is the bursting of the sac; this may also occur subcutaneously: the fluid is poured out into the subcutaneous cellular tissue, and there becomes absorbed, or a diffuse inflammation of the cellular tissue occurs. The most unusual termination is the rupture of both the sac and the skin; the further course is, then, the same as in an incised or punctured wound of the bursa, of which we have spoken already.

More frequent than the form commencing acutely is that which is *chronic* from the beginning. It arises entirely without pain, very slowly, more frequently in elderly than in very young people. In England this chronic dropsy of the bursa præpatellaris has received the name of "housemaid's knee." It is said to be very common there in female servants, who do a good deal of work in a kneeling position. To me it appears somewhat doubtful, however, whether this can have any influence upon the origin of the disease, since several anatomists have pointed out that in the kneeling position it is not the patella but the condyles of the tibia upon which the body rests. To touch the ground with the anterior surface of the patella it would be necessary to lie almost entirely upon one's face, but it is possible that the great tension of the skin over the patella in much kneeling may be an occasional cause of this disease.

As regards the contents of these dropsical sacs, they are *very much thinner* than in the sheaths of the tendons; but these sacs also not unfrequently contain fibrinous corpuscles which, on palpation of the sac with the fingers, rub against each other and produce a sensation

similar to that of starch. The sac itself becomes much thickened in the course of time.

In *acute* cases the following treatment should be adopted: above all things the patient must remain quiet in bed; you should then paint the sac freely and repeatedly with tincture of iodine. The dropsy generally disappears rapidly, and you may attempt to get rid of the last remains thereof by compression by means of bandages or strips of adhesive plaster; or you may employ pressure from the first with wet bandages, or envelope the knee in a hydropathic wrapping. Mercurial plaster or ointment also does good service.

Chronic dropsy of the bursa præpatellaris causes so little inconvenience that it does not in general come under the surgeon's notice until late. Most of the patients are scarcely incommoded at all thereby in walking; others state that they feel fatigue in that limb sooner than formerly. The disease is, for the most part, confined to one side, but may be double. It is extremely difficult to effect absorption of chronic dropsy of this bursa by means of the remedies mentioned above. If any attempt is to be made to remove the evil it must be of an operative character. A simple puncture is of no more use here in the long run than in other dropsies, because fresh fluid collects; the puncture, to be effectual, must be followed by the injection of tincture of iodine. This is free from danger if the patient remains at rest during the treatment, and a radical cure generally follows. Another mode of treatment is the laying open of the sac, which is followed by suppuration. If the sac is very thick we are justified in extirpating it altogether, but this must always be done with great caution, to avoid injury to the adjacent capsule of the joint. Volkmann has recommended a mode of treatment which I have several times adopted with great success, namely, forcible compression: a padded, bent splint of wood or metal, as broad as the hand, is placed behind the knee, which is to be pressed against it as firmly as possible with very strong flannel bandages; this mode of compression, which is generally followed by œdema of the foot and frequently by violent pain, must be continued for several days. Absorption is effected in small hygromata in two to three days, in large, very old ones in six to eight days. I have seen very good results from this method in hygroma præpatellare; it is less certain in hydrops genu, and seldom of any use in dropsy of the sheaths of the tendons.

LECTURE XXXIX.

C. CHRONIC RHEUMATIC INFLAMMATION OF THE JOINTS. ARTHRITIS DEFORMANS. MALUM SENILE COXÆ.—*Anatomy. Different forms. Symptoms. Diagnosis. Prognosis. Treatment.* APPENDIX I: ON LOOSE BODIES IN THE JOINTS: 1. *Fibrinous bodies.* 2. *Cartilaginous and bony bodies. Symptomatology. Operations.*—APPENDIX II.: *On neuroses of the joints.*

C. *Chronic rheumatic infiltration of the joints. Chronic rheumatism of the joints. Arthrite sêche. Rheumatic gout. Arthritis deformans. Chondritis hyperplastica tuberosa. Malum senile coxæ.*

You will start back with affright at this long array of names, all of which denote the same process of disease anatomically, and may fairly ask why so many names should be used for the same thing. When a disease has received so many names, this is often a sign either that its nature is not yet thoroughly understood, or has been very differently comprehended at different times; this is, however, by no means the case here, but the process itself has always been comprehended in the same manner anatomically, and all the investigators are fully agreed as to the results. The best plan will be to begin with the anatomy. The disease very especially affects the cartilage, secondarily also the synovial membrane, as well as the periosteum and the bone; in most cases the affection of the cartilage is no doubt the primary one. The changes which we meet with in the cartilage are the following: it becomes uneven at single points, then rough on the surface, and in advanced stages disappears entirely here and there, and leaves the bone exposed in places quite smooth and, as it were, polished.

If you examine the eroded cartilage you will find its cavities enlarged and containing cells on the point of division; these cells

are not, however, so small nor so little developed as is otherwise the case in cell-formations accompanying inflammations, but are well developed and partly recognisable as fresh cartilage-cells; the process is an infinitely slow one, and the newly formed cells attain a higher degree of development (Fig. 109) than in the earlier-described form of inflammation (Fig. 104); softening of the intercellular tissue does not follow here as otherwise in inflammation, but a separation of the fibres. The peculiarity of the process is already characterised thereby, but it includes much more that is remarkable.

Fig. 109.

Degeneration of the cartilage in arthritis deformans; at *a* fatty degeneration of the cartilage-cells. Magnified 350 times, according to O. Weber.

The roughened cartilage does not resist the friction of the articular extremities of the bones against each other; it gradually becomes rubbed away, and disappears from this friction as far as the bone. Immediately beneath the cartilage there always lies a layer of a tolerably compact bony substance, very thin it is true, next to which comes at once the spongy extremity of the epiphysis;

after the destruction of the cartilage, the friction next affects this layer, and in it fresh bone substance is formed in consequence of the mechanical irritation produced thereby; the medulla of the spongy substance ossifies over a slight extent at the point where the friction occurs. In spite of this, the opposed bones gradually become more and more worn away through the movements in the joint, but since the friction at the same time constantly gives rise to a fresh formation of bone substance, the surface so rubbed generally remains firm and smooth, because the process of sclerosis always precedes the loss from friction. It may thus gradually happen, if the joint remains movable, that a considerable portion of the bone becomes rubbed away, and that the very defective articular extremity of the bone still continues smooth. These polished surfaces are met with in the hip, on the upper surface of the head of the femur, and on the acetabulum; in the knee, on both the condyles, &c. The spongy substance of the neck of the femur may, during this process, become at some points porous, while sclerosis results at the points of friction; the neck of the femur may, at the same time, become covered with osteophytes, and thereby assume a very extraordinary shape. This process will appear to you extremely peculiar: at one point, wasting of bone; at another, new formation of bone during the same process, in close proximity in the same bone! The disease not unfrequently commences as an uneven exuberant growth of bone, and terminates in atrophy of bone! I think, however, that you must already be accustomed to this combination of wasting and new formation in chronic inflammatory processes; if you do but recall to mind caries, or the process of ulceration in general, we have there also seen decay on the surface of the ulcer and new formation in the vicinity on an extensive scale.

To the already described affections of the cartilage and the bone are to be added certain changes in the synovial membrane, which do not differ much, however, from those met with in chronic dropsy of the joints; the cavity of the joint contains synovia only little increased in quantity, but turbid, thin, and mixed with the particles of cartilage detached by friction. The membrane itself is thickened, but slightly vascular, and only the villi, which are frequently much elongated, are furnished with an increase of vascular loops at their extremities. But *the parts about the joint* may also participate in the disease: the periosteum, the tendons, and the muscles. In

these, ossification sometimes takes place very gradually, so that the articular extremities of the bones become covered thickly externally with a newly formed bony mass; these exuberant growths of bone sometimes attain a very great development. The form of these osteophytes is entirely different from that with which you are already acquainted; they are here smooth, roundish, and not shaped like pointed stalactites, but rather appear as if cast; they are, moreover, not so porous as other osteophytes, but consist in all their layers of more compact bony substance, and are for the most part covered with a thin layer of cartilage, which is not the case with other osteophytes. This form of joint-disease is already so strongly characterised externally by these peculiarities, which you will easily comprehend on examining a series of preparations, that we can very easily recognise them, even in macerated preparations of bone, without knowing anything of the case in question (see Figs. 110, 111, 112).

Why the new formation of the bone in this disease assumes such

FIG. 110.

FIGS. 110 and 112.—Osteophytes in commencing arthritis deformans. FIG. 110, lower end of humerus. Reduced in size. *a*. Osteophytes; *b*. polished surface of the bone.

FIG. 111.

FIG. 111.— Carious elbow-joint, fungous inflammation of the joint, osteophytes resembling stalactites. [Reduced in size.

FIG. 112.

FIG. 112.—Os metacarpi I. *a* and *b*. as in Fig. 110.

an entirely peculiar character depends probably, on the one hand, upon the slow process of development, on the other, upon the circumstance that the ossification is not here preceded by any especially copious vascularity, as is the case with the osteophytes which are formed in the healing of fractures, in caries, necrosis, ostitis, &c. If a tissue is highly vascular before it becomes ossified, a porous bone substance must be formed, for the greater the number of vessels so many more cavities are there in the bone. But in arthritis deformans no considerable new formation of vessels precedes the formation of bone, the tissues become converted into cartilage and bone just as they are: *the periosteum, tendons, even the capsule of the joint, the ligaments and muscles,* and all this takes place extremely slowly. It thus happens that denser bone is formed. It also sometimes happens here that, in the midst of the subserous cellular tissue in the vicinity of the bone, entirely isolated bony points arise, which remain for a long time as detached, round pieces; these do not unite perhaps until late with the rest of the bony mass, and then appear as if glued on, so that we can often trace in the form of the new growth of bone the manner of its formation. The articular extremities of the bones may be completely displaced by these periarticular new formations of bone, and assume a perfectly abnormal, half dislocated position. *The joint may thereby become quite immovable,* while, with slight formation of such new growths and a high degree of friction-atrophy, it may be very abnormally movable. In many cases, these formations of bone grow *into the joint,* become detached there, and form loose bodies in the joint, of which more later on. Lastly, I have to remark that chronic dropsy may also supervene, and you will easily understand that with all these concurring circumstances, the joints may become so deformed that the disease has been justly named "arthritis deformans." *I must here repeat, however, that none of all these pathological changes ever lead to suppuration.*

We now come to the *clinical characters* of this peculiar disease. According to my experience, three forms are distinguishable; one which is for the most part polyarticular and combined with contractions of the muscles, a second which occurs in a single joint in youth and middle age, and a third which occurs in old age only. Combinations of these forms with each other are also met with.

1. *Polyarticular chronic rheumatism* ("arthrite sêche, rheumatismus nodosus, rheumatic gout, rheumatische Gicht") occurs in

persons of middle age and in young people, more frequently in women than in men, in poor people than in rich. Ill-nourished, anæmic individuals are especially liable thereto, although the disease occasionally becomes developed in very stout women. Acute articular rheumatism, or a gonorrhœal inflammation of the joints, may form the starting-point, but frequently no cause whatever can be discovered. After the acute or subacute stage of these joint-affections has passed off, there often remains in individual joints, most commonly in the knees, stiffness, pain, and slight swelling. But the disease may also commence quite gradually in a chronic form, with irregular, moderate pain in the joints. The patients use their extremities quite well at first; in the course of months and years, however, the power of motion gradually decreases greatly; after exertion or exposure to cold, intercurrent, subacute dropsies of the joints occur, but a portion of the effused fluid becomes re-absorbed; the joint, however, always remains a little stiffer after each exacerbation and sometimes thicker also. In the morning, when the patients get up, their limbs are so stiff that they cannot be moved at all; after some preparatory movements, things go on better during the day, but towards evening the joints become painful again. A fresh symptom now gradually appears; the muscles dwindle away, the legs remain in a flexed position. The wasting muscles are exceedingly prone to contract, and this tendency is much favoured by the joints remaining fixed in one attitude. The general condition of the patients is good, the appetite and digestion unimpaired; they may even gain flesh, and only suffer from hectic fever during an exacerbation of an acute attack of the joint-mischief. And so it may go on for a year or more. At last the muscles almost completely waste away, and the joints become deformed and stiff. The patients are, to use a vulgar expression, "all contracted." When the disease attacks the hip or knee, they are completely bed-ridden; nevertheless, with skilful and appropriate nursing, they may be kept alive for a year or more. The joints most frequently attacked are the knee-, hip-, wrist-, finger-, ankle-, and shoulder-joints.

2. *Arthritis deformans* is nearly always confined to one joint; it seldom appears in two corresponding joints, and attacks persons who were in other respects perfectly strong and healthy. I have seen it more frequently in men than women. This form has obtained its name from the deformity which is caused by the bony

growths from the articular periosteum and the wearing away of the cartilage. My experience of it has been confined to a few cases in the hip, one case in both knees, two cases in the shoulder, once in the foot, and once in the elbow. As a rule there is no obvious exciting cause, although some cases were preceded by dislocations or sprains. The joints in this affection are usually painless, stiff, and at the same time œdematous. They often contain loose bony substances, and the synovial membrane may be completely lined with fatty cysts.

3. *Malum senile coxæ.*—When this disease attacks aged people it assumes a milder form than in some bad forms of chronic rheumatism. The hip is the most common seat of the disease, whence the name "Malum senile coxæ," yet old people suffer not unfrequently from this affection in the shoulder, knee, and elbow, and still more often in the fingers and great toes.

The onset is usually very gradual, and accompanied with but little pain. The symptoms are often those of sciatica, but with more stiffness, and rarely with any acute initial stage. The stiffness, which is especially troublesome in the morning, is frequently the only symptom complained of in the beginning of the disease. When the joint is used in walking its function is slowly regained; the grating in the joint, however, is often so marked that the patient himself draws the attention of the surgeon to it. In those cases where the morbid process has originated in the fingers, acute attacks with severe pain and some febrile disturbance are especially seen; moreover, in the course of a year the affected joints are quite deformed and thickened. The big toe undergoes spontaneous dislocation, and the head of the os metatarsi primum, thickened with bony exostosis, stands out alone. When the hip is involved lameness is an early symptom; the exostoses are usually unimportant in old people, although the thigh shortens, and the head of the femur and the acetabulum are worn away. The muscles rapidly atrophy, and in the course of a few years the hip joint becomes firmly anchylosed. The disease is much more common among men than among women, and especially among those who are badly nourished. Rigidity of the arteries, ossification of the ribs, with the intervertebral joints, with ossification of the anterior vertebral ligament, are results which are often found in patients affected with *malum senile coxæ* in more than one joint. The diagnosis of *malum senile coxæ* is clear, and if the description which I have

given you is borne in mind, you will not easily fall into any error of diagnosis.

When the above disease attacks young people in one joint, it is sometimes doubtful at first whether we have to deal with a granulating inflammation of the joint or with arthritis deformans, but any doubts are easily eliminated by further observation of the case. Further confusion may arise in later stages between fungous inflammation of a joint and caries sicca, in both of which diseases there is atrophy of the muscles and friction in the joint, gradually supervening in young and otherwise healthy subjects. In caries sicca, however, the bony outgrowths are not so generally spread through the joint as in arthritis deformans, which even when very chronic never shows any disposition to ulcerate, and is very much less painful. But when the chronic rheumatic joint inflammation is bilateral, or appears simultaneously in several joints, and when to this is added the reflex contraction of the muscles, caused by the irritation of the synovial membrane, then indeed there is no longer any doubt as to the nature of the disease. Rheumatismus nodosus is often confounded with gout, presenting, as it does, somewhat similar appearances in the hands and feet. Gout differs from it in respect to its specific attacks, and the marked difference in the acidity of the urine, which sufficiently characterise it as a perfectly distinct disease; and to this point I have already drawn your attention.

The prognosis of rheumatism affecting several joints is, from a therapeutic point of view, very unfavorable. Indeed, I look upon it as being incurable in old people. In young people, by dint of careful and patient treatment the progress of the disease may be arrested at a certain point, and some slight amelioration may ensue, but even this trifling result is obtained with difficulty, and a perfect cure is rarely achieved. The explanation of these unsatisfactory results lies in the anatomical changes produced by the disease; the loss of bone and cartilage is not made good, and the new bony growth is not absorbed; it is much too firm and solid for this. There is no natural action of the muscles to aid in their nutrition, for they are so enfeebled as scarcely to be able to move the fixed and unwieldy joints. In the treatment of such a patient you will have need of much perseverance, nor must you be surprised to find him consulting one surgeon after another, and even placing himself under the care of some bone-setter; nay, he may at last reproach

you with being the *fons et origo* of his long and troublesome illness.

They depend rather upon some disposition to morbid change in the heart and arteries, and sclerotic changes taking place in internal organs.

We must treat these patients to the best of our ability, we cannot choose only the promising cases; the incurable and the dying have their claim on us, and where we cannot stay the disease, we must at least use our every effort to alleviate pain and suffering. By its simultaneous appearance in different joints, chronic rheumatic arthritis shows that it arises not from some local injury affecting a particular joint from without; but in most cases from some general morbid condition. The rheumatic diathesis, complicated as it is with a tendency to inflammations of serous membranes and exudations in joint and muscles, is often set down as being at the root of the matter, and hence we resort to anti-rheumatic remedies. The prolonged use of iodide of potassium, colchicum and aconite, diaphoretics and diuretics, is recommended, though their administration is followed with but small results; still there are no other remedies at our command which have any specific effect on rheumatism. The internal administration of Carlsbad waters has been in some cases attended with beneficial results, both in this disease and in simple gout.

In addition to these and other remedies which are required by the idiosyncrasy of the individual patient, warm baths, and especially the hot springs of Wildbad in Würtemberg, Wilbadgastein in Salzkammergut, Wiesbaden, Baden near Zurich, Ragatz in St. Gallen, Baden-Baden, Teplitz in Bohemia, Krapina Teplitz in Croatia, Mehadia in Hungary, have been recommended. Besides these, salt baths may be used, especially the less irritating ones, in cases of incipient muscular atrophy. Great discrimination must be used with regard to the climate of the watering place, inasmuch as all these patients are peculiarly sensitive to a moist atmosphere and sudden falls of temperature. The hot sulphur springs must be used with care and discontinued on the appearance of a subacute exacerbation. If such patients live in localities where cold and wet winters prevail they should winter in Italy, at Nice, Pisa, or Palermo, where the houses are well-built and able to keep out the cold weather. Damp houses must especially be avoided. The patients must keep themselves warm, and habitually wear woollen clothing;

and the affected joints should always be wrapped in flannel. Hydropathic treatment has been much recommended, and has been followed by some success. Certainly when it is rationally pursued under medical supervision, and not left to the discretion of proprietors of hydropathic establishments, good results have followed. The patients become hardier, and more capable of resisting changes of temperature. The copious draughts of water, and the wrapping up after the baths, act in two ways, namely, diaphoretically and diuretically; lastly, a good moral effect is obtained by the patient conscientiously submitting to the rules laid down for him, and substituting a hygienic treatment for one based on the use of medicines alone. It is well known that patients at hydropathic establishments often take great interest in their cure, and exhibit no small amount of gratitude even where the results are infinitesimal. When the general condition of the patient is not too enfeebled, and he has not an insurmountable aversion, as sometimes happens, to the treatment, it is advisable to try it, and if any advantage is to be derived therefrom, it must be continued for not less than a year. The Russian vapour baths have in some cases been followed by good results.

In ill-nourished subjects the disease has yielded under cod-liver oil, quinine, and iron. As regards local treatment, various kinds of inunction, in which friction takes its share, can be used, such as iodine ointment, purified fat, ammoniated liniment, and so on. The more powerful derivative drugs are not of much value here; even tincture of iodine is only useful in subacute attacks, where also blisters are worth a trial. You must be cautious in the employment of all-powerful irritants to the joints. Douches are often most valuable in some very chronic and tedious cases; warm douches, vapour douches, and local sulphur baths, mud and "moor" baths, have proved of service in some cases; while in others the softest rain douches falling from only the height of a foot act as excitants. We cannot always foretel the effect of the baths; this must be noted by the patient himself, under the guidance of the physician. If pain supervenes, the douches may be abandoned for a time, after which the patient may carefully resume them. If the pain again returns with increased vigour, the douches must then be discarded altogether. Now, shall we keep the joints entirely at rest or allow some degree of movement to take place? Complete rest is unadvisable for several reasons: the joints become perfectly stiff, and

that in some unfavorable position; and what is more, the absolute rest favours in a high degree the already advancing atrophy of the muscles. Moderate motion, passive as well as active, should be allowed, but never to such an extent as to produce fatigue or great pain. The patient himself can manage the passive motion; or, what is better still, he can use a most ingenious instrument constructed by Bonnet for this purpose. Lastly, one word about the atrophy of the muscles; we can endeavour to strengthen the muscles by friction, electricity and regulated motion, partly active, partly passive; hygenic gymnastics are not altogether useless towards the attainment of this object. I have seen the most happy results follow a judicious course of gymnastics, and varied frictions, under the superintendence of Dr. Barbieri. All treatment of this kind must be persevered in for months and even years, in order that the full benefit may be derived therefrom.

You will see from the foregoing therapeutic review that we are not poor in resources, which can be had recourse to with success in the treatment of chronic rheumatism. Unfortunately, all these means of treatment are costly, tedious, and quite beyond the reach of the poor, who chiefly suffer from the malady in question. For in the huts of the poor, dry warm air, good food, shelter from draughts, and baths, are for the most part unattainable; and without them, forming, as they do, the very foundation of the treatment, the administration of expensive drugs is only so much money thrown away. As I have already remarked, the earlier these patients come under treatment, and the younger they are, the better. Under these circumstances you may succeed in arresting the progress of the disease. When it has reached a certain stage, it is difficult enough to arrest the disease, and, of course, a cure is out of the question. Although the use of the above-named remedies in conjunction with warm spring baths may do much to ameliorate the condition of the patient in Malum coxæ senile, I regard it for the most part as an incurable complaint. Arthritis deformans affecting one joint is incurable when the joint gives too much trouble; it can be removed by resection or amputation.

Appendix I.

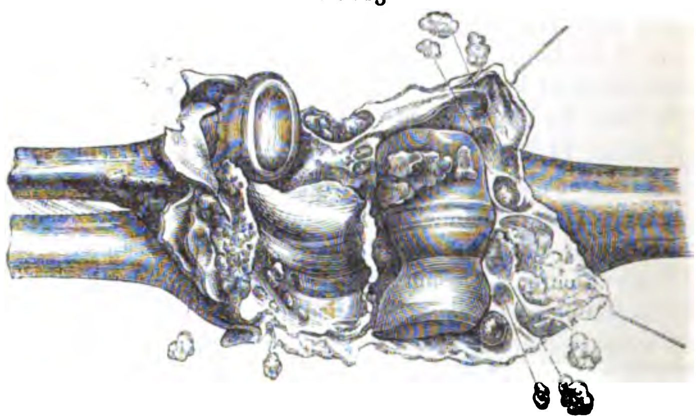
On loose bodies in joints. Mures articulares.

By loose bodies in joints we understand the presence of more or less compact bodies originating in a joint. Foreign bodies entering the joint from without, such as a needle, bullet, &c., or detached piece of bone, lying loose in the joint, are not comprised in this category. There are two varieties of loose bodies in joints: (1) small, oval irregular bodies of a melon-seed shape, which are developed in large numbers, and which are seen under the microscope to consist of fibrine. They originate in joints with chronic effusion, and are deposits from the increased and altered nature of the synovial fluid, resembling the similar bodies in effusions into the sheaths of tendons. It is possible that such bodies may arise from extravasations of blood. This variety of loose body gives no occasion in itself for operative interference, but is merely an accidental complication of chronic effusion into a joint. Sometimes their presence can be diagnosed by the soft grating which can be detected in such cases on palpation of the joint. However, this complication in no way demands any modification of the treatment already laid down in chronic effusion into joints, and only complicates it in so far as the increased difficulty added in the reduction of the joint to its normal circumference. (2.) The other variety of loose bodies in joints consists of bones always covered with a thin layer of cartilage, and sometimes adherent, at other times loose in the joint. Their shape is varied, often quite fantastic; the term "joint mouse" seems to have arisen from an accidental resemblance to a mouse. These bodies are seldom uniformly oval or round, but often ragged and warty just like the osteophytes in arthritis deformans.

Examined microscopically, they are found to consist of a thin layer of true fibrous cartilage containing an ossified central point, which is sometimes only calcified. These bodies then, inasmuch as they consist of organised tissue and not of deposits from the synovial fluid, even when found loose, must have originated from the same structures as they are composed of and become detached at a later period. Such is indeed the case; they are, for the most part, osteophytes which have penetrated into the joint from without; they rarely arise from the folds of the synovial membrane. The villi sometimes contain normal cartilage cells, which may begin to

proliferate, and in this way cartilaginous granules, tumours, or enchondromata may be developed, and, later on, become ossified. These excrescences may remain attached to the villi for a time, but finally become detached and float freely in the joint. The most common variety, however, of loose bodies in joints is that which developes close under the capsule of the joint in the synovial membrane. This consists of bony cartilaginous bodies, osteophylites, which get twisted and finally torn off and become loose in the joints, Apparently, when once detached, they are incapable of further growth, but can be nourished by the surrounding synovial fluid. Amalide is of opinion that these bodies are always in the first place bony, and that the cartilaginous covering is a secondary deposit.

FIG. 113.



Loose bodies in the elbow-joint (after Cruveilhier). A very rare case. These bodies usually occur in the knee-joint.

Associated with the development of loose bodies in joints there always exists a certain amount of effusion into the joint, and this is possibly in some cases the primary disease. These bodies appear almost exclusively, or at least chiefly, in the knee-joint, and are confined to adults; they are extremely rare, perhaps they constitute the rarest joint disease. There exists an undoubted connection between the loose bodies found in arthritis deformans and hydrarthrosis; they belong to the same class of disease, and give rise to an acquired or hereditary diathesis depending upon the opposition to fungous and suppurative fungous inflammation of joints. The symptoms, which may be regarded as pathognomonic of loose bodies in joints, are the following: the patient suffers for some time from

considerable effusion into the joint, and possibly is unaware of it; all of a sudden while walking he feels a sharp sudden pain, which for the moment prevents him from going any further. The knee then remains fixed in a half flexed or extended position, and is only able to be moved again after being rubbed and manipulated. This phenomenon is caused by the loose bodies getting jammed between the articular surfaces of the knee-joint, between the inter-articular cartilages, or in a synovial pouch. But for some time before this jamming occurs, perhaps weeks or months, the patients may complain of weakness or slight pain in the knee, and an examination will reveal, as has been already mentioned, some slight degree of effusion into the joint. The patients not unfrequently, from the way in which the violent pain comes on and disappears again, themselves come to the conclusion that there is a movable body in the joint, and frequently can feel it plainly themselves; sometimes they are able by certain movements of the joint to enable the surgeon to grasp it. In other cases the surgeon is the first to feel it after repeated examinations, and is able to push it about in different directions. Very often it may vanish for several days or weeks before taking up a position in which it may be felt from without. All these symptoms are only well marked when the body is detached; as long as it still remains adherent, or is too large to be jammed, it causes but little or no trouble. When the pain of a loose body in the joint and a moderate amount of effusion are not continuously severe, and do not exhibit any tendency to further spontaneous development, there may be no suppurative inflammation, but only from time to time a subacute inflammation with serous effusion. In other cases, however, the pain caused by the jamming is every moment so agonising that many persons attacked with it ask for relief at any cost. Attempts to fix these bodies by exciting adhesive inflammation by means of compress bandages, tincture of iodine or vesicants, have not met with much success. The operation consists in the extraction of the loose body, and is performed as follows: the loose body is firmly pressed up under the skin on one side of the joint, the skin is then still further stretched, and is then cut through into the capsule upon the body, which is allowed to slip out, or is raised through the incision by means of an ear-spoon such as is made by Fock. The wound is closed with the finger, the leg is extended, and the skin is allowed to slip back into its natural position, so that the cut lies deep in the capsule, and the

two wounds do not directly communicate with each other. The wound in the skin is then closed with plaster, and the extended joint placed in a splint. A plaster bandage can be applied with advantage, which can be prepared with a large window and adjusted before the operation. The subsequent appearance of inflammation is to be treated in the same way as a traumatic joint inflammation. Formerly this operation was attended with very unsatisfactory results, not unfrequently severe inflammation set in, and the surgeons were fortunate if the life of the patient was saved after amputation of the limb above the joint. Various operations have been had recourse to, but the one just described is the most simple, and is now generally adopted. Fock has carried out this method of operating five times with perfect success. The subsequent inflammation was trifling, and most of the patients were back at their business in a few weeks after the operation. Just as in removal of a cataract, or in cutting for stone, it is essential to the success of the operation, that it goes smoothly and without any material obstacle or impediment from bleeding. When the loose bodies cause no uneasiness, it is sufficient to apply a knee-cap, in order to reduce the effusion and to supply a certain degree of firmness, so that no unwonted movement may be made with it. The patient often derives much comfort from this treatment.

Appendix II.

On neurosis of joints.

By neurosis and neuralgia are understood those diseases which reveal themselves by attacks of pain, which are sometimes intense and characteristic; and whose origin is not to be traced to any change in the tissues.

We conjecture from this a functional disturbance in the nerves without any morphological change. That there is a purely functional disturbance which we are in the habit of describing as weakness and hyperæsthesia occurring in the tissues, and especially in the nerves, which we are unable, even with the aid of all the modern means of research to detect either any morphological or chemical change during life or after death, is beyond a doubt. Whether such a change does exist, which we are unable to detect, we are unable to say, for that which we cannot perceive with our

senses has no real existence for us. We thus call those conditions of the joints, in which there are pains but no morbid process can be found, joint-neuroses.

The typical neuroses do not appear at certain times in the day as in neuralgia of the nervus trigeminus. Brodie was the first who differentiated joint neuroses as a distinct class of disease; Esmarch, Stromeyer, and Wernher have more recently studied these conditions and have further clinically developed their diagnosis. In the opinion of these authors, there are those diseases to be taken into consideration which are attended with some slight anatomical changes and accompanied by painful sensations and disturbed function, and which in relation to their severity are out of all proportion to the slight degree of disease which they present. Joint neuroses must be placed in the category of sensitive, psychical, hyperæsthesia, with their reflex complications. In short, for the most part they belong to that class of psychic disease known as hysteria and hypochondriasis. These cases which I have seen in my own practice, and those described by the above authors as joint neuroses, I formerly regarded, partly as slight diseases of the joints the symptoms of which were exaggerated by hysterical women and girls, and in some cases simulated, partly as commencing and unrecognisable joint and bone diseases, and lastly, partly as increased sensitiveness which had remained after the disappearance of the original disease. It is quite possible to give a name to this group of diseases, but they are not to be looked at from one point of view only, nor all to be treated on one plan. General medical experience and knowledge of men must help most in the treatment of hysteria; no one would believe in the caprice and consequence of simulating contractions and cramps, except an experienced medical man.

Hysteria is essentially a mental disease often either incurable or only partially so. In treating the hyperæsthesia of the joints, the cold-water cure, douches, and sea baths are useful, with regular exercise of the joint, as recommended especially by Esmarch. I have seen the best results obtained in such cases from hot springs, baths, and electricity. Shampooing is often most beneficial in joint neuroses.

LECTURE XL.

ON ANKYLOSIS; VARIETIES; ANATOMICAL CONDITIONS; DIAGNOSIS; TREATMENT; GRADUAL, FORCED EXTENSION, OPERATION.

CHAPTER XVIII.

On Ankylosis.

THAT by ankylosis (from ἀγκύλος, curve) is meant a stiff joint you already know. I must, however, add that this term is only to be applied to those cases where the acute or chronic process of the disease which gave rise to the stiffness has passed off and partial or complete immobility of the limb is the only symptom remaining. If, during an inflammation of the knee or hip-joints, an extremely flexed position of the joint is kept up owing to an involuntary long continued contraction of the muscle, and so on account of the pain the joint cannot be moved although there is no mechanical obstacle present, we do not speak of it as an ankylosis, but as an inflammation of the joint with contraction of the muscles. The reason a joint is incapable of being extended even when the process of inflammation has subsided is to be found in a mechanical obstruction outside, sometimes inside, and sometimes in the substance of the joint itself. A muscle contracted from atrophy and shrinking or a contracted cicatrix, especially if it be on the flexed aspect, will materially impair the mobility of otherwise healthy joints. These causes should be borne in mind when speaking briefly of ankylosis of this or that joint, and it should be described as muscle or cicatricial contraction. If we wish to describe this restricted mobility as an ankylosis we can do so, provided that we are careful to distinguish it as an ankylosis depending on causes extraneous to the joint and call it spurious ankylosis and so on. There only now remain those cases of stiff joints which result from pathological changes in

essential parts of the joints. Here we have to deal with the following conditions:

1. Cicatricial growths between the opposed surfaces of the joint itself. These vary exceedingly in their nature and numbers. They originate after the healing of the fungous inflammation of the joint in the proliferating growth of granulating tissue. Hence, band-like adhesions form like those seen between the pulmonary and costal pleura, or tightly stretched bands of lymph.

FIG. 114.

Fibrous bands in a resection of the elbow-joint from an adult, almost natural size.

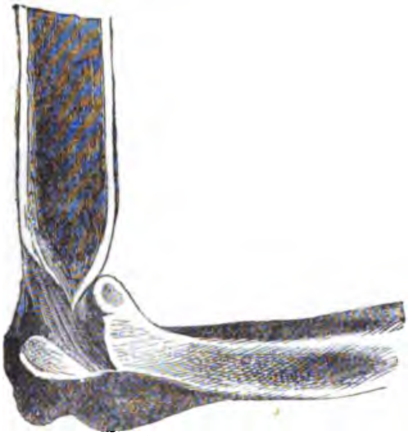
By this means the cartilage may be partly preserved, but usually the layer of cartilage and part of the bone are destroyed. These growths ordinarily consist of fibrous tissue like other cicatricial structures (Fig. 115). In many cases this fibrous tissue ossifies and the two ends of the joint are bridged over with bony tissue or the entire surfaces may become blended together (Fig. 116).

2. Other impediments to mobility are cicatricial contraction of the capsule of the joint and the accessory ligaments, as well as the interarticular cartilages which may be entirely destroyed. These cicatricial contractions not only appear in those spots where fistulæ had formed, but also without suppuration, whilst each tissue, which has been more or less weakened by long plastic infiltration, has, later on, after the inflammatory process has ceased, a tendency to shrink.

3. A not unimportant obstacle to mobility, and sometimes the reason why, after fungous inflammation of a joint of a protracted nature, motion is never restored, is caused by the necessary shrinking and destruction of the synovial sac lining the joint. In order to make this clear to you, I must briefly draw your attention to the condition of the larger joints when in motion. The capsule of the

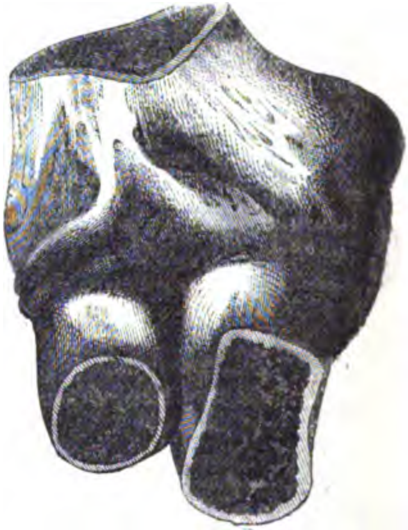
joint is never sufficiently elastic to allow of its adapting itself to all the movements of the joint without stretching. Imagine the humerus flexed on the thorax; in that case the capsule underneath the joint would be greatly contracted, and above just as tightly

FIG. 115.



Complete cicatricial growth over the surfaces of the elbow-joint of a child; the trochlea humeri and part of the olecranon are destroyed. Longitudinal section. Natural size.

FIG. 116.



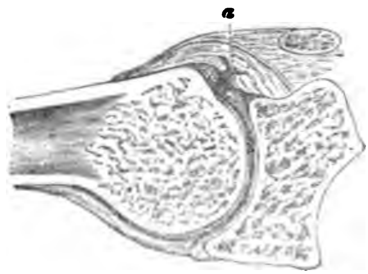
Bony ankylosis of elbow-joint of an adult. Nearly natural size.

stretched out. Again, imagine the arm raised high; in that case the upper part of the capsule would be very much contracted and the under part very much stretched; the joint capsule ought to be as elastic as india rubber. This, however, is by no means the case. The capsule is either not contracted at all at the different extremities of the joint, or but very little; it is folded in distinct, regular lines. Should the position of the head of the joint become changed the folds are opened out again, and on the opposite side, where it was smooth before, a new fold of the capsule is formed. In Fig. 117 is a section of the anterior surface (anterior section after Henle) of the shoulder-joint in a raised position, and in Fig. 118 a section of it in a depressed position. If the synovial membrane becomes diseased the joint usually remains in one position; the humerus is generally depressed, and thus the synovial pouch suppurates and becomes cicatrised and deformed underneath (Fig. 118*a*). Even when the

joint is uninjured above it is no longer possible to raise the arm on account of the inability to expand of the capsule at the under surface of the joint. Ankyloses thus originate in the already existing layer of cartilage; the secretion of synovial fluid ceases. Lastly, the cartilage may in the course of years degenerate into fibrous tissue (as in old unreduced dislocations), or become ossified, and so set up an immovable ankylosis. Similar conditions exist in all joints; the best representations are to be found in 'Henle's Anatomy.' Volk-

FIG. 117.
The capsule folded above.

FIG. 118.
The capsule folded underneath.



Anterior sections at the shoulder-joint.

mann formerly described this variety of ankylosis, which especially attacks young persons after subacute coxitis (arising out of rheumatic and puerperal inflammations of joints) without ulceration, but with marked contraction of the muscles, under the title of "cartilaginous ankylosis." The term is an appropriate one, because the cartilage remains unchanged for a lengthened period.

4. A further mechanical impediment may arise in the exostoses which develope themselves round the joint outside the head of the affected bones. According as the olecranon or coronoid depression of the humerus is implicated with new bony growth, so is either the coronoid process or the olecranon process of the above unable to lock. In the former case it cannot be fully flexed, in the latter it cannot be fully extended. This impediment arises espeeially in arthritis deformans, seldom in fungous inflammation of a joint (Fig. 110).

5. Lastly, such mischief may arise as a result of caries of the heads of the bone that the epiphyses are quite unevenly opposed to each other and can no longer be replaced. Their surfaces are too

much altered, and no longer fit one another, and in this abnormal position (pathological dislocation) are no longer capable of being moved towards one another. Look back once again to Fig. 115. In consequence of the destruction of the trochlea humeri the ulna is fixed to the humerus, so that although a slight movement can be made perfect flexion is impossible on account of the coronoid process of the ulna being fixed to the humerus and the coronoid depression of the humerus having disappeared. In like manner the tibia in caries of the knee may be displaced outwards and backwards, the condyles of the femur remaining free, appear to develop, so that the surfaces of the joint are no longer capable of interlocking.

In addition to those causes of ankylosis which depend more or less on some abnormal conditions within the joint, there are others which act from without. These are contractions of muscles or cicatrices which develop simultaneously with the degeneration of tendons and bones, and contribute in a large degree to the fixing of the joint in a false position. Also degeneration and fixing of the inner surfaces of tendons are likely to produce stiffness and immobility. This may take place in the hand after long-standing inflammation without suppuration having taken place in the sheaths of the tendons. All the fingers are stiff and immovable, generally extended, and the joints intact. Skilful manipulation with passive motion may act like magic in overcoming this stiffness, and the fingers may again be brought into use.

The diagnosis of ankylosis is easy enough, but it is not so easy to determine which of the above-mentioned conditions is the cause of partial or complete immobility of the joint. In cases of complete immobility bony ankylosis is likely to be diagnosed, but it is not always the condition; complete immobility may also result from adhesions and wide fibrous growths. The longer such an ankylosis exists the greater is the likelihood of its becoming osseous even when the joint is but slightly diseased and when the cartilages are nearly whole, for if the joint remains at rest for years, perhaps only on account of contraction of the capsule, a perfect bony ankylosis must result. A perfectly healthy joint would in the course of time, if kept at rest, become ankylosed. Experimental researches show, according to Menzel, that in a joint kept at rest for some time the following changes take place: a proliferation of the cartilage cells is set up which leads to a granulating metamorphosis of the covering of the cartilage whilst the secretion

of the synovial fluid ceases altogether. For a healthy condition of the synovial membrane and the cartilage movement is indispensable. And so we see that, in those joints such as the intervertebral, pelvic, and sternal joints where little or no motion is allowed of, there is a very rudimentary synovial membrane and extremely stunted growth of cartilage. We have dwelt upon this in order to show what conclusions as regards the mobility of a joint may be drawn from a knowledge of the duration of the ankylosis. When the ankylosis is even only slightly movable the synovial membrane is seldom entirely destroyed, and in such cases there may even remain a small portion of the cartilage. It is easy to fall into error with regard to the mobility or immobility of an ankylosis unless attention is paid to the amount of elasticity remaining in the muscles after the termination of an inflammation in a joint. It is often impossible to form a trustworthy opinion before a complete examination has been made under chloroform.

What then is to be done in these ankyloses? Is it possible to restore mobility to stiff joints? In most cases, yes. Can this mobility be retained as well as the normal functions restored? Unfortunately, this is seldom possible; as a rule it is impossible. What will happen then? of what use is any treatment? The last question is in some cases a pertinent one, but not so in the majority. We have already repeatedly mentioned the fact that as a rule in joint inflammations the limbs assume unfavorable positions in the late stages. A leg fixed at right angles at the knee is an undesirable and useless burden, and formerly it was the practice to amputate such limbs, because the patients could get about better on a wooden leg than by means of two crutches. An arm completely extended or only slightly flexed at the elbow-joint is for purposes of seizing and moving objects a perfectly useless limb. By bringing the ankylosed limbs into positions which are relatively the most useful, such as extension of hip- and knee-joints, flexion at right angles of the elbow-joint, much good may be done for the patients. Hence operations for straightening or bending ankyloses are of the greatest value. Ankyloses in unfavorable positions have been very common for a long time; they will become fewer and will eventually disappear when once the principle, for which we have so strongly contended: the early treatment of the joints when in the stages of acute or chronic inflammation, is pursued with a view to securing the most favorable position in the event of anky-

losis finally taking place. It will seldom happen to a surgeon now-a-days to be called on to perform operations for the improvement of the position of an ankylosed limb which he has treated during inflammation of the joint.

Still there are always a large number of cases in the country which must be treated under unfavorable conditions, and in which there is angular ankylosis in the knee- and hip-joints, so that the operations for straightening ankyloses are not unfrequent.

The attempts to reduce crooked limbs are not by any means new. We find drawings and descriptions, in the writings of surgeons in the middle ages, of instruments designed for this purpose, for the method of reduction by continual extension with machines is the oldest. A large quantity of apparatus has been constructed for the different joints, by the aid of which extension and flexion of the extremities may be effected with screws.

This form of apparatus is still chiefly employed for the purpose of straightening the limb and at the same time preserving its mobility, but inasmuch as these results are more readily attained by instantaneous extension it has fallen into disuse.

In competition with the slow gradual extension is the rapid violent extension, or the falsely so-called "brisement forcé."

This operation, before chloroform was used, was beset with many difficulties; it was extremely painful and not free from danger, great force had to be used in violently breaking down and tearing through the ankylosis, and this force was rendered still more necessary on account of the powerful contractions in the muscles caused by the pain. It was often necessary to divide the tendons of the contracted muscles before the ankylosis could be broken down, thus complicating the operation. The after-treatment was but imperfectly understood; the limbs were put into splints or fastened firmly in apparatus. Acute inflammation and considerable swelling were the results, and so the method met with no general approval. Bouvier and Dieffenbach were almost the only surgeons who resorted to this method. Other surgeons preferred regarding these cases as incurable, or contented themselves with sending them to be treated by gradual extension by orthopædists. When the patients were poor people amputation was resorted to, on the ground that they could follow their occupations better with a wooden leg. Such was the condition of things when B. v. Langenbeck, 1846, made the first experiments in breaking down ankylosis at the knee under

chloroform. The result was the discovery of the extremely interesting fact, at that time unknown, that under the influence of chloroform the contracted muscles become quite lax and flexible, and can be stretched like india rubber. This obviated the necessity for dividing tendons and muscles in these operations. As the operation was painless under chloroform, it could be performed slowly and carefully by manipulation alone. The results were so unusually favorable that this method, hardly deserving in its new form the somewhat brutal title of "brisement forcé," came into general use, and for some time has supplanted, perhaps too completely, the more gradual extension by instruments and weights.

The method of operating, the indications for doing so, the after-treatment, and the precautions to be taken, were so carefully worked out by B. v. Langenbeck himself, that this operation may now be regarded as one of the safest and simplest there is. In order that you may not conceive too dreadful an idea of this operation on account of its name, "brisement forcé," I will describe the operation of forced extension of a knee ankylosed at right angles. The patient is first placed on his back and is gradually anæsthetised until all the musclcs are perfectly flaccid, and there is complete absence of reflex contraction on motion; the patient is then turned over on to his stomach, an assistant then holds his head, while another places his arm under the patient's breast to allow of his breathing; the pulse and respiration are carefully watched so that the operation may be abandoned on the appearance of any dangerous symptoms of too deep anæsthesia. The patient is then drawn in the prone position down the table until his knee rests on the edge of the table, to which a well-stuffed horsehair cushion is firmly attached. An assistant now leans all his weight with both hands on the thigh; the operator stands on the outside at the left knee and places his left hand in the popliteal space so as to press on the thigh; with his right hand he seizes the leg just below the condyles of the tibia, and then with his right hand forcibly presses outwards the bent-up knee. If the ankylosis is still recent and not too immovable, the leg will yield little by little, with a faint crackling and tearing, into a perfectly straight position. If this manœuvre is not readily effected, the operator must seize the leg lower down, somewhere on the calf or just below it. If this is done, however, too much violence must be avoided, for in this position, where there is a certain weakness of the bones, the tibia might easily

be fractured below the condyles; the force must be chiefly spent in movements of extension. Should the method just described prove unsuccessful, we must endeavour to break down the adhesions of the joint by forcible flexion; the leg is grasped from the front, and efforts are made by slow and uniform pressure to flex it. By this means the adhesions are more easily broken down than by the extension movement. When once some of the adhesions are broken down extension becomes easy enough. All painful wrenching and pushing is decidedly dangerous, and scarcely ever attains its object. If the degree of extension obtained is considered sufficient for one operation, or the leg is completely extended, the patient is placed on his back and the knee is fastened firmly down by means of Hueter's bandages. The leg is firmly extended at the foot, and a plaster-of-Paris bandage is applied from the foot upwards to within an inch of the perineum. A thick layer of cotton wool is previously laid round the knee and extended upwards and downwards to each end of the plaster-of-Paris bandage, where the greatest pressure takes place later on. But as the plaster of Paris is not always hardened by the time that the patient has recovered from the anæsthesia, it is advisable to apply a splint well padded along the back of the leg to prevent the knee from becoming flexed again. The splint may be removed in about three or four hours afterwards, by which time the bandage will be firm enough to withstand the contractions of the muscles. The pain which the patient feels in the joint on recovery from the chloroform is not always very severe; in proportion to the force used it is trifling. The foot swells and becomes œdematous occasionally when it has not been properly bandaged; when this is the case, or when it happens immediately after the operation, no further complication arises. If the pains are severe after the operation an ice-bag may be applied over the bandage and a subcutaneous injection of morphia administered. In eight or ten days' time we may yield to the wishes of the patient, and allow him to get about with a stick or crutches. After eight or twelve weeks the ankylosis has healed in the new position, the patient has meanwhile thrown aside the crutches and walks with a stick or wholly unsupported, and if the knee is stiff it is at all events straight. The bandage can now be removed, and the patient may be regarded as cured.

In the case just described we have taken it for granted that one operation was sufficient to effect a complete extension of the knee.

This, however, is not the rule; in many cases we do not always venture so far in the first operation for fear of causing injuries which might be followed by serious consequences. What then are the conditions which contraindicate the completion of the operation at one sitting? We must be especially cautious in cases of tense cicatrices in the skin; cicatrices at the bend of the knee are often difficult to stretch, and are liable to rupture under forced extension. The cicatrices sometimes lie over the larger trunks of vessels and nerves which may have been implicated during the early stages of ulceration; a laceration of these structures might prove a most dangerous and even fatal complication. Suppuration and ulceration may result after laceration of a cicatrix, and we must, therefore, be careful not to stretch a cicatricial tissue to its utmost limit. When during extension the cicatrices have become very tense, we must desist and apply the bandages, and repeat the operation after an interval of from four to five weeks, and so on, until the extension is complete. Another condition necessitating cautious procedure is displacement of the tibia, which may arise during the course of caries of the knee, when it is especially prone to dislocation backwards. Under any circumstances it is difficult and in some cases impossible to remedy this position of the tibia; gradual extension answers best in such cases. Forced extension in such a case would be followed by complete dislocation backwards, and thus effectually preclude the possibility of straightening the joint.

You must not expect that these knees, even when completely extended, will resume their natural beautiful shape; that is never the case. It may happen that even after the most perfect and complete extension has been effected the leg still remains too short, because from the first onset of the disease development had been arrested. But as we are not obliged, like the Scotch, to go about with bare knees, the shape of the knee is not of much importance compared with its straightness and strength. When joints affected with white swelling, or with fistulæ, can be brought at once into the most favorable position for use and placed in a closed or capsule bandage, even then the time when the fistula is but just closed and the cicatrices recent, brawny, and easily lacerated, is a most unfavourable one for attemping extension, for at this time laceration of the cicatrised skin on fresh suppuration may easily occur. In such cases I never attempt extension under chloroform, but

invariably resort to extension by means of weights. What has here been said with regard to ankylosis of the knee applies equally, not to dilate upon the subject, to ankyloses of the hip and foot. Ankyloses of the foot and shoulder are of an altogether different importance as regards their functions; with them we are chiefly concerned in the restoration of mobility, and this is not to be achieved by breaking down the ankylosis and applying a plaster-of-Paris bandage. If the object, after the breaking down of an ankylosed knee, be to obtain an ameliorated state of the joint with mobility thereof, we must obviously remove the plaster-of-Paris bandage soon after the operation, otherwise we should be compelled to resort to apparatus for restoring the extension and mobility, or the employment of manipulation. I do not deny that there are cases in which a certain mobility may be produced by these means; but they are of rare occurrence, and happen chiefly where there has been a fracture of a joint, or a short transitory inflammation leaving a certain degree of stiffness behind. I am inclined to believe that in the former case mobility may have become established in the course of time by daily use. After rheumatic and puerperal inflammation of joints it is undoubtedly most important that the stretched, but in the first place not very firm growths, especially those of the synovial pouches, should be broken down at the right moment after the subsidence of the inflammation. Later on, not only do the new growths become firmer, but the shrunken ligaments become less yielding, and the cartilages degenerate and atrophy, or ossify. We should not indulge in too many illusions with regard to the results of extension in ankylosis. It is already a great advance in surgery to be able almost to eliminate ankylosis from the list of diseases in which amputation is indicated. The way to the attainment of still more brilliant results is by no means barred. In ankyloses, which still admit of some degree of motion, extension by means of apparatus or weights can always be tried in the first instance. There is no doubt but that as improvements in this direction are introduced the use of forced extension will diminish. There are cases in which the mechanical conditions in the joints present an insuperable obstacle to their being brought into any other position. I have already quoted the case of an elbow joint as an example; first there is an arthritis deformans, then the cavities at the lower end of the humerus are filled with new bony growth, rendering it impossible to move the ulna either backwards or for-

wards. Similar changes occur as a result of arthritis deformans in other joints. The ankyloses resulting from these changes are just as difficult to produce motion in as those resulting from true arthritis. In both these diseases, then, the breaking down of the ankylosis is contraindicated. Lastly, as already mentioned, these growths in the joints may result in bony ankylosis, which can seldom, except where there are but one or two bony bridges, be broken down. In most of these cases the ankylosis remains firm and cannot be broken down. What is to be done in such a case? The position of the joint in such a case may be changed in two ways: bending in the bone either above or below the joint or by excising a portion of the joint or bone. With regard to the first mode, many surgeons would demur to its being advocated as a method, and yet this bending in of the bone even so as to fracture, for example, the inner or outer condyle of the femur in extension of the knee-joint, has often been done accidentally and generally with good results.

It has several times happened to me, in extending an ankylosed knee, and once in an ankylosis of the hip, to partially or even completely fracture the bone involuntarily. The joint itself remained *in statu quo;* at the knee the bone was bent above the joint at an angle which compensated for the abnormal position of joint, and at the hip the bone was bent in a similar way below the joint; in this way the limbs were straightened although not by breaking down of the ankyloses. In all these cases I applied the plaster-of-Paris bandage; the result was the same as always obtains in a simple subcutaneous fracture, the pain less than after rupture of an ankylosis, and the final results most satisfactory. I do not at all see why this method of successfully treating intractable ankyloses by fracture of the bone should not be decidedly preferred to resection of the hip or knee, especially where it can be performed without any considerable violence or unjustifiable force. I am, indeed, of opinion that subcutaneous fracture should always be preferred to resection of the knee, seeing that it is such a simple operation. In other joints, however, resection is to be preferred on different grounds.

There are three different methods of resection in bony ankylosis:

1. Rhea Barton (this method was known in 1825), in cases of angular knee ankylosis after a preliminary division with the saw,

cuts out a wedge-shaped piece of bone, just above the joint. The base of the wedge is above and its angle lying underneath corresponds to a certain extent with the angle of the ankylosis. This wedge could just as well be removed from the joint itself. The limb is then straightened, the joint remaining intact. The deformity is thus overcome in the thigh, as in the operation of subcutaneous fracture. This operation has been followed by good results in ankylosis of the hip and knee.

2. We can resort to B. v. Langenbeck's operation of subcutaneous osteotomy through the ankylosed joint. This procedure, which we have already pointed out as of value in crookedly united fractures, and rickets (see pages 254 and 565), has at present received but little attention and so it can scarcely be criticised. Gross has obtained very successful results from a modification of it which consists in drilling diagonally through the ankylosis and then disuniting it with small chisels.

3. Complete resection of the joint.—I have already given my opinion on the propriety of resection in ankylosis of the hip and knee, and should only regard it as an ultimum remedium and valde anceps.

Resection has been strongly advocated for the removal of ankylosis of the elbow-joint, and by means of this operation *if all goes well*, we are able to make a false joint which is tolerably useful. The result, however, is not certain and we cannot always control it. Who would risk his life to get rid of a stiff elbow-joint? The results after resection of ankylosed elbow-joints have not always been successful either with regard to the mobility of the joint or quoad vitam, although brilliant results may appear to have been obtained for a short time. We must not, however, dilate further upon the question of resection. With regard to the shoulder-joint we have a peculiar condition to deal with. Experience has shown that persons with stiff shoulders do by continually moving the shoulder-blade make it so movable as to obviate any necessity for operating in such cases. Patients with caries of the wrist are generally so thankful when the disease after many years heals up that they are not at all eager to complain about any stiffness which may result in the hand. However successful resections of wrist-joints have of late years been carried out, but as yet nothing is known about the final results. With regard to the foot, resection in cases of unfavorable positions is out of the question; the

defect usually arises in the tarsal bones which gives rise to deformity after inflammation of the joints. It will depend entirely upon the nature of each individual case whether the foot will be useful for locomotion, or whether extension could be obtained, or lastly whether a good healthy stump after amputation would not be preferable.

LECTURE XLI.

CHAPTER XIX.

ON CONGENITAL MYO- AND NEUROPATHIC JOINT DEFORMITIES AND ON CICATRICIAL CONTRACTIONS. LOXARTHROSES.[1]

I. Deformities of embryonic origin, arising from arrested development of the joints. II. Deformities only arising in children and young people through arrested growth of the joints. III. Deformities depending upon contraction or paralysis of several muscular groups of muscles. IV. Diminished movement of joints, resulting from contraction of fascia and ligaments. V. Cicatricial contractions. Therapeutics: Extension by apparatus. Extension during anæsthesia. Compression. Tenotomy and myotomy. Division of fascia and ligaments. Gymnastics. Electricity. Artificial muscles. Instrumental support.

Gentlemen,—We have to consider to-day those deformities which do not always originate in primary diseases of the joints, but which depend on abnormal mechanical conditions of the joints; it may be that the articular surfaces undergo abnormal changes from different causes, or that in normal joints the mobility is destroyed in one or more directions through some impediment resulting from some lesion of the muscles, fascia, tendons, or skin. We have to deal chiefly with stiffness, deformity, and impaired mobility in joints, which are outside the synovial sacs. I follow in this section the classification adopted by Volkmann, whose excellent work on this subject, in his book on surgery, edited by V. Pitha and myself, I cannot sufficiently commend to your study.

I. *Deformities of embryonic origin, arising from arrested development of the joints.*

These distortions are always congenital; they are more frequently

[1] Loxarthrosis, from λόξος, crooked, ἄρθρον, joint.

found in the foot than elsewhere, often under the form of so-called "club-foot, Pes varus, equino-varus." In those cases of distorted feet which are called "club" feet, the condition which is usually seen is that deformity in which the inner edge of the foot is raised. The plantar surface of the foot is usually flexed, and in children can either not at all or only with great difficulty be brought by the hand into its normal position. When patients born with this deformity, which is chiefly bilateral, learn to walk they tread on the outer edge of the foot. This gradually twists more and more inwards and becomes more or less broad. The sole of the foot contracts, the middle and fore parts of the foot no longer develop, the joints of the tarsal bones become ankylosed, and finally the feet reach the stage of deformed club-feet. The outsides of the insteps become flattened, and thickened callosities are formed underneath which bursæ are developed. From this position the foot cannot be moved, the muscles of the leg atrophy, and hardly any structures but skin and bone remain, and thus a resemblance to a horse's hoof is originated. We have to differentiate various stages of club-foot, beginning from slight and unimportant deformities arising immediately after birth down to the more advanced stages just described. It is noteworthy that the advanced stages of club-foot only result from using the feet in walking; were such a patient never to use his feet the congenital deformity would most likely undergo but little or no change. With regard to the etiology of congenital club-foot the most contradictory theories have been advanced. The typical and nearly always constant condition in congenital distortions would seem to point to some typical disturbance in development of the lower extremities; for if it depended upon fœtal diseases, disturbances of an irritative nature or undue pressure on the fœtus there would be well-marked differences between the cases which we should recognise later. In my opinion investigations of the highest value have recently been made with regard to these malformations. Eschricht has shown that the lower extremities at the beginning of their development grew from the abdominal surfaces of the embryo, and that their posterior surfaces and the heads of the knee are turned in an axial twisting towards the abdomen. The outgrowths of the extremities lie so close together that they appear actually to be fused together under one membrane, and thus the above-mentioned axial twisting cannot take place, and in such deformities (sirens) the feet are always directed backwards. This type restrained

by the above conditions stands in relation especially to that class of congenital club-foot in which the axial twisting of the feet is not fully accomplished.

Congenital club-foot, therefore, comes under the category of arrested development. With regard to the causation of this arrested development we are able at present to give just as little explanation as of other deformities of this kind. As consequences of the faulty position in which the foot remains in the uterus, and in which later on it grows, are the abnormal lengths of the muscles, of which the gastrocnemius is the most remarkable and best known example, and the deformities especially seen in the tarsal bones to which Hueter has drawn attention. These entirely new and minute observations on the etiology of congenital club-foot are so clearly in advance of the purely hypothetical causes such as myelitis in utero causing paralysis and contraction of the muscles as to relegate the latter to the domain of history. A few other distortions of the feet depend on known abnormal growths or conditions of undue pressure. Volkmann has collected some highly interesting observations on this point; these cases are all, however, different in some respect from each other—a proof that they depend upon manifold accidents. In other cases large bones have not developed at all as the lower end of the tibia or fibula; the lower end or the whole of the radius (manus vara). In the vertebral column several halves of the cartilages on one side do not develop, or are overdeveloped and result in a bending on one side of the vertebral column (scoliosis). These cases of congenital scoliosis are, nevertheless, not altogether unknown; the Vienna Museum contains several such examples of scoliosis. Lastly, we must mention here the incomplete development of the sterno-cleido-mastoid muscle which is not uncommon as a congenital deformity and furnishes an apparently typical form. The vertebræ are, as far as is known, normal in this condition, and but little is known regarding the causation of this distortion. The hypotheses advanced on the subject seem to me to have no likelihood whatever of being correct ones.

II. *Deformities only arising in children and young people through arrested growth of the joints.*

All movements of the body such as walking, standing, sitting, &c., are performed partly by means of the form of the joints and

their ligaments and partly by the action of the muscles. You will at once recognise the importance of the muscles in all our positions, even those of rest, when you attempt to place a dead body, the muscles of which are no longer contractile, in a given position. You will then see that the action of the muscles is of far more importance than the shape of the joints or their ligaments. Those people whose muscles are easily fatigued either because they are ill-developed, or weakened by some illness, or are not exercised or never used through laziness, seek those positions for them in which they need not be brought into activity. The articular pressure which is continually distributed in all directions by the action of the muscles in the joint undergoes a harmless alteration through the disuse of the muscles, and in this way only one part of the joint bears the whole pressure. These abnormal pressures have no ill consequences when they do not last long and when the bones are fully developed. But when bones which are still growing and are weak and likely to remain so for some time are attacked, are kept in one position for any length of time and subjected to pressure at one point, the shape of surfaces of the joint and the ligaments gradually changes. The bones become implicated secondarily; inflammation is set up by the pressure, and a pathological condition is established which is often accompanied with pain, and quickly leads to destruction of the normal tissues of the joint. The ligaments and muscles adapt themselves and the changes begun on one side of the body react through physical laws on the form and development of the whole skeleton. As most important instances of this class I cite scoliosis, genu valgum, and pes planum.

By "scoliosis" (from σκολιός, curved) is understood that condition of the spine in which it has gradually become curved to one side to such a degree that permanent curvature is produced. We have already noticed the fact that this may arise from abnormal development of the intervertebral cartilages; it may also result from excessive pressure caused by pleuritic effusion, or after contraction and falling in of one side of the thorax after absorption or emptying of the exudation, or from a fixing of the pelvis in one position whether as a result of one leg being shorter than the other after joint or bone mischief or other causes. All these changes are comparatively seldom the cause of the scolioses which we are now considering and which are especially prone to develop in young girls just before puberty. This class of scoliosis is almost typical;

as a rule, the lumbar region is convex towards the left and the dorsal region convex towards the right. It is disputed as to whether the lower or upper curvature arises first, whether the first is the primary and the second the secondary or compensatory change, or *vice versa*. As a rule both conditions are found together from the beginning, and appear to originate simultaneously.

When the faulty position remains unnoticed or untreated and the destructive process goes on, the right shoulder-blade is raised, and this is the first remarkable symptom; the intervertebral cartilages gradually sink in, and the deformity becomes worse and worse. The upper part of the spine is bent forwards, the position of the head is changed, the patient becomes, in a word, humpbacked, as you must have seen them. As H. Meyer has pointed out, the outward curvature is developed backwards for anatomical reasons, and so we term this distortion "Kypho Scoliosis" (from κῦφος humpback). Most old people with humped backs, whom you see, belong to this class. Patients with caries of the spine seldom live to be old. The so-called quart-pot humpback resulting from caries only occurs in children and young people. The chief causes of scoliosis is weakness of the muscles of the back or inertia. As long as weakly children are left to themselves, and can lie down, sit, walk and run as they like, and as much as they like, scoliosis does not usually develop itself, but as soon as they are restrained in one position for hours and fatigued by writing, reading, sewing, playing the piano, &c., they assume during their occupations those positions in which the muscles are the least used in maintaining the upright posture, and these constrained attitudes become constant. When the children sit down without anything in front to lean on, they rest with one hand on the seat; when they stand they bend forwards so that the upper part of the body is not kept erect; oftentimes they stand on one leg to allow the other one to rest, &c. When once the curvature of the spine has remained for months or years, the centre of gravity of the trunk and head is altered, and the curvature rapidly progresses.

At the commencement only the intervertebral ligaments are compressed on one side, then those on the other side are implicated and become thickened; next the intervertebral cartilages become pressed on one side until a wedge shape is formed. This compression sometimes leads to inflammatory new growths of the nature of osteophytes, and occasionally ossification of the ligaments.

Genu valgum, baker's knee, is that deformity of the knee-joint in which the leg is in such relation to the knee-joint as to form an obtuse angle on the outside with the thigh. If these persons lie on their backs with the knees together the feet are widely separated; in order for them to allow the inner borders of their feet to touch they are obliged to cross their knees. This deformity is most common in young men who are obliged to stand all day long working hard with their bodies and arms and pressing down on their knees; bakers, locksmiths, and cabinet makers are chiefly attacked by these deformities of a higher degree and more rapid course accompanied with great pain in the joints. Gradually the external condyle is pressed in, the internal lateral ligament firmly stretched, the external lateral ligament contracted, the biceps shortened and contracted.

Flat foot, Pes planus, is a not uncommon distortion of the foot, which is more often seen in young girls than boys just before puberty, especially when they are compelled to stand too much. The bones which form the arch of the foot, along its inner border, sink in so that the instep becomes flat, then the outer border of the foot (pes valgus) is raised and the perinei muscles, whose points of attachment are approached, become shortened. This deformity is very common and may result as a consequence of genu valgum or rickety distortion of the leg; it more frequently arises alone, however, and sometimes rapidly and with severe pains. Although I fully recognise long-continued pressure on growing bones as a cause of scoliosis, genu valgum, and pes planus, still the practical observations which I have made in these cases are that they do not entirely depend on the above causes, but that there is present a weakness of the muscles and the bones themselves. I cannot but help thinking that some slight tendency to rachitis has something to do with it.

This explanation has been brought forward by many authors, especially by Lorinser. The idea that the articular surfaces become deformed and uneven has been advocated by Hueter and Henke, and certainly plays a considerable part in the growth of these deformities, although it can hardly be accepted as a primary cause. That the contraction and relaxation of the ligaments of the joints cause these deformities as an idiopathic process, as I was formerly inclined to believe, appears, from the more recent investigations, to be improbable, seeing that they are invariably

present in displacements and deformities of the heads of the bones.

III. *Deformities depending on contractions or paralysis of several muscles or groups of muscles.*

The number of cases which belong to this class is not very great. Acute inflammatory processes which have their seat in the muscles or the neighbouring tissues under strong fasciæ cause contraction, on account of the severe pain in the inflamed muscles. In deep abscesses of the neck it is usual for the head to be bent towards the diseased side, and the patient is unable, even by the exercise of all his will and strength, to straighten it; this can only be effected under complete anæsthesia. I have seen a foot fixed in the position of pes equinus by an abscess which had developed in the calf of the leg. Acute inflammation of the psoas muscle (Psoitis with peri-psoitis) is followed by a flexed position of the hip-joint at an acute angle. As the pus drains off the contraction diminishes, and often disappears entirely; sometimes, however, the cicatrix from the abscess is so extensive that, as it heals up, the contraction becomes very firm, and later on it is extremely difficult to move. Next in frequency, direct nervous irritation, through disease of the nervous centres, gives rise to permanent contraction; these cases, when they depend on centric nervous disturbance, offer the most remote chances of amelioration by means of therapeutics. In caries of the spine and inflammation of the anterior columns of the spinal cord, contractions and paralyses of the muscles may occur; in one case of this kind I have seen a spontaneous cure effected. Further, reflex contraction may be a cause; I have seen this in the hip, hand, and foot, especially in young females; these contractions were in some cases complicated by falls on the affected part or by hysteria (see "Joint Neuroses"). They are recognised by their cessation during sleep and under chloroform. Finally, we come to the most frequent variety of these cases, the so-called *paralytic contractions*, which are especially developed in partial or complete paralysis after meningitis and encephalitis, more especially in children.

Essential paralysis of children.—These contractions occur nearly always on one or both sides of the lower extremities. A completely lame leg hangs or remains in any position in which it is placed by the mechanical conditions appertaining to it; this you can verify on any dead body. If the foot is not moved from this position, the

distortion gradually becomes permanent, so that the posterior ligaments of the foot, the muscles of the calf, the tendo Achilles, and the fasciæ covering them, partly contract and partly thicken. Gradually the articular surfaces and the shape of the bones are destroyed by the uneven pressure and become more distorted; finally, the foot will probably be permanently deformed. The examination is obscured by the opposition offered by the muscles and tendons, and this gave rise to the opinion that the gastrocnemius was contracted in cases in which it was paralysed with the other muscles of the leg. It was thought that the extensor muscles must have been completely paralysed, and that the antagonistic muscles were innervated, so that they alone acted on the foot. In this way the theory of Delpech was originated regarding antagonistic contractions, which were especially marked in cases of disturbed equilibrium from paresis and paralysis of particular groups of muscles.

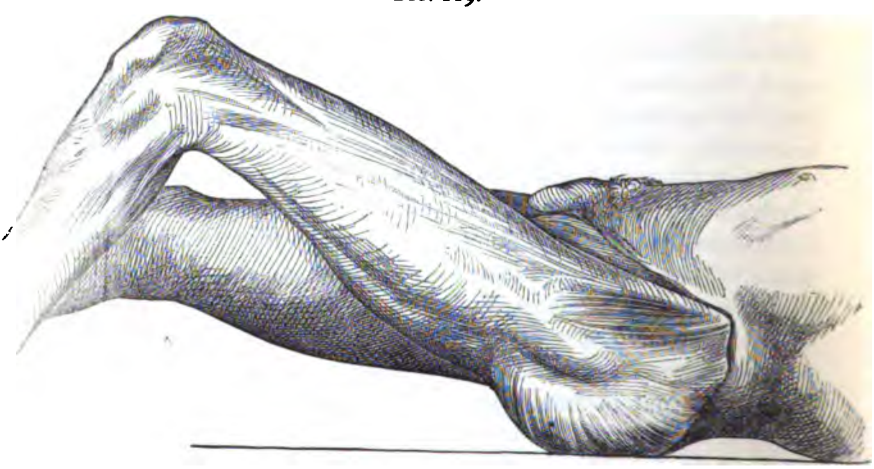
Hueter first drew attention to the fact that permanent position of the paralysed joints, attributed to contractions and the so-called antagonistic contractions, did not depend on active contraction of the muscles, but, as in congenital club-foot, from disuse, arrested growth, and atrophy. I have often met with cases which made me sceptical with regard to the theory of antagonistic contractions; the following case is a good example:—A soldier at the battle of Sadowa was shot through the right forearm, and the radial nerve was lacerated; four years later there was total paralysis of all the parts supplied by the radial nerve, but there was no sign of antagonistic contraction. If we pursue our investigations on paralysed limbs still further we shall find that, in those cases in which patients sit all day with the hip flexed and the knee flexed and hanging down, contractions in a flexed position are formed in the hip- and knee-joints. When the patients are strong enough to move about with crutches, the immobility of the joint is reached more slowly. The foot, when placed on the ground and overloaded by the weight of the body, gives way outwards (*pes plano-valgus paralyticus*), the knee bends out in front (*genu antecurvatum*), the trunk is bent forwards over the hip until it is supported by the sound leg or a crutch. In this way, according to Volkmann, vicious positions of joints are gradually caused by the pressure of the superincumbent body, and in young people marked changes of the articular surfaces thus originate. All these conditions are most rationally explained on mechanical principles; formerly the most complicated theories

with regard to actual momentum were elaborated, but they are misleading.

IV. *Impaired mobility of joints caused by contractions of fasciæ and ligaments.*

Any long-continued fixed position of a limb, even if, as in the above cases, it does not depend on diseases of the nerves or muscles, may result in contraction of the fascia. A man who had suffered for a year and a half from suppuration of the inguinal glands had, during that time, lain with the left hip and knee flexed; when the bubo was healed he was brought to our clinique because he was unable to straighten his leg. The fascia lata, if allowed to remain in a state of rest for several months, becomes so rigid that it is impossible to stretch it out again. At the termination of a coxitis, when the joint has become completely healed, this contraction of the leg may become a permanent impediment to its extension, and the patient may suffer from a crooked limb for the remainder of his life. This is a new and important reason for the practice of keeping the limb in an appropriate position whilst the inflammation is running its course.

FIG. 119.



Shrinking of the fascia lata in coxitis. After Froriep.

V. *Cicatricial contractions.*

Cicatricial contractions have already been mentioned; they result from the fact that the inflammatory new growths in the wound give up by degrees their fluids, while the original gelatinous richly vascular granulation material gradually contracts into dry connective tissue, and at the same time, by being strongly drawn together, the obliteration of most of the blood-vessels is effected.

FIG. 120.

FIG. 121.

Cicatricial contractions after burning.

The larger the area involved by the scar, the stronger in all directions are the contractions. All wounds with widespread destruction of skin are followed by extensive cicatricial contractions, and while large tracts of skin are seldom so frequently destroyed as after burns, the scars resulting from burns are always accompanied by the most marked deformities. It naturally depends to a great extent on the position of the cicatrix whether or not it is followed by contraction or distortion. Scars on the flexed surface of a joint extending far in the long axis of the limb may entirely prevent the limb from being extended. Contracted cicatrices on the neck are followed by drawing down and immobility of the head on the

affected side (Fig. 120). Scars of the cheek may draw down the mouth and lower eyelid. Scars of the hand and foot in the neighbourhood of the finger joints often give rise to distortion and partial immobility of the joints (Fig. 121). Scars of deeper tissues, such as the muscles and tendons, give rise, as already mentioned, to easily recognised deformities. Necrosis of the tendons is easily excited by injury to them, and they are replaced by cicatricial tissue, which gives rise to a permanent distortion and stiffness, as for example, in a finger.

The diagnosis of the conditions need not be dwelt upon, inasmuch as it is quite clear when the etiology and origin of these deformities are understood. As regards the prognosis in these distortions, everything depends on whether their cause can be removed, and on this will the treatment in different cases depend.

The first thing we aim at in the removal of contractions is the attempt to extend the contracted parts; we may try to effect this by stretching the contracted limbs several times daily. These manœuvres, or manipulation as they are called, require great care and skill, and it seems more convenient to effect them by means of the more regular action of some apparatus. The extension apparatus, which is still employed, consists of a combination of a screw and tooth-wheel, a mechanism which has been used in surgery from time immemorial. The apparatuses are constructed in a variety of ways; they must, however, be light, firm, and well padded. Such apparatus is easily constructed for the knee- and elbow-joints; for the shoulder- and hip-joints, however, they are unsuited, as it is extremely awkward to adjust them to the shoulder-blade and pelvis. Extension under chloroform may be resorted to from time to time to help things on a little quicker, but all violent stretching must be avoided, for the contracted muscles are very inelastic, and only permit of being very gradually stretched. In those contractions of muscles depending upon neuroses mechanical modes of extension are hardly advisable, and can, at the most, only be used as supplemental means of treatment; the chief attention must be directed to the nerve lesion, which is the cause of the contraction. In contractions of ligaments and fasciæ, the extension treatment by means of instruments (orthopædics, from ὀρθός, straight, and παιδεία, education) is especially resorted to. In the place of these machines plaster-of-Paris bandages and permanent extension have been much resorted to in many cases during the last ten years, and the treatment of these

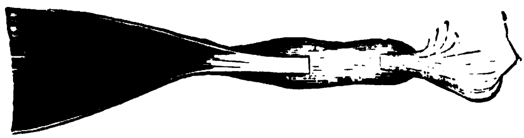
cases has thus been much simplified in the hands of practical surgeons. I must, however, reserve my judgment on the respective clinical advantages of these methods in individual cases. Cicatricial contractions can be somewhat improved by stretching of the cicatrices, although seldom perfectly cured. The extension in cases of cicatricial contraction is kept up by means of bandages and compresses. By these means, in the course of a year spontaneous atrophy takes place in the cicatrices. The stretching of the cicatrices is effected by compression, as in the treatment of annular cicatricial narrowings of canals, so-called strictures, which are frequent in the urethra and œsophagus, by means of elastic sounds called bougies, because they were formerly made of wax, of different sizes. The orthopædic methods of treatment hitherto discussed do not always attain their object, and then only slowly. Cutting tendons of contracted muscles, or the division of the muscles themselves, was practised in the middle ages. These operations are called "Tenotomy and Myotomy" (from τένων, tendon, μῦς, muscle, τέμνω, to cut); the first-named operation is much more frequently resorted to than the last. The operation was formerly performed in the following manner: the skin was divided down to the tendon, which was then cut through, and the wound allowed to heal by suppuration. The results were seldom brilliant. The suppuration was often profuse; thickened cicatrices were developed which had to be gradually stretched out. It was Stromeyer who first raised the operation to the rank of a successful proceeding by resorting to the subcutaneous section of the tendons. Dieffenbach elaborated this method, and it is now universally resorted to in practice. I will first briefly describe the operation before proceeding to discuss the results obtained by it. Let us take as an example the tendon which is most frequently divided—the tendo Achillis. The best instrument to use is Dieffenbach's tenotome, which resembles a slightly curved, pointed penknife. The patient is placed on his stomach, and the leg is held by an assistant, the foot is grasped by the left hand of the operator, the tenotomy knife is held in the right hand. The operator pierces the skin near the tendon, and brings it down over the tendon, taking care not to pierce the skin a second time. The blade of the knife is then turned on to the extended tendon, and the tendon is cut through with a crisp sound. The moment the tendon is divided a sensation of recoil is experienced in the left hand, and the foot can be slightly moved.

The knife is then carefully withdrawn. The wound caused by the entrance of the knife is now all that can be seen; the division of the tendon has been completely effected under the skin. The mode of operating just described of dividing the tendon from without inwards is the easiest for beginners, inasmuch as there is no danger of making a second wound in the skin. A more elegant and suitable mode in many cases is to divide the tendon from within outwards. The position is the same as in the other operation: the skin is pierced in the same way, but the knife is carried underneath the tendon, and the edge of the knife is directed against the tendon, the thumb of the cutting hand is placed against the projecting point of the knife to control its depth and to feel that it does not pierce the skin. The knife is then pressed and drawn from within outwards through the tendon. Care must be taken not to stretch the foot too tightly, lest the skin might be cut through by the start of the knife when it has divided the tendon. This method appears to be more difficult than it really is; however, it is necessary to practise it on the cadaver.

When the tendon is divided there is as a rule very little bleeding from the external wound; there may be, however, considerable hæmorrhage from the division of a large branch of the posterior tibial artery, which lies underneath the tendon. Where there is slight bleeding it is sufficient to apply a small piece of English plaster, which can be fixed with collodium. Where the hæmorrhage is more profuse a small compress is applied, and fastened with a bandage applied round the foot and up as far as the calf of the leg; this invariably arrests the hæmorrhage. The bandage can be removed after twenty-four hours, and plaster applied. The wound nearly always heals by first intention; after three or four days the puncture is closed. Suppuration may, however, supervene, the whole region of the wound becomes red, swollen, and irritable, an ichorous discharge escapes from the puncture. An abscess forms on the opposite side, which must be opened, and, when the suppuration is not attended by any dangerous symptoms, it may go on for two or three weeks, and materially endanger the success of the operation. If it continues longer, the thickened cicatrices resulting must be gradually extended. Immediately after the operation a depression can be felt at the point of division, caused by retraction of the muscle after the division of the tendon. This depression disappears after twenty-four hours, and is replaced in a few days by

a swelling which generally decreases, and, after fourteen days at the longest, the tendon appears to be perfectly reunited. The steps of this healing process have been experimentally studied. It was formerly regarded as a perfect kind of regeneration. I have repeatedly made these experiments on animals, and find that the healing process is similar to that in nerves and bones. When the tendon is cut through and the muscle has retracted, an empty space is left at the point of division unless the surrounding cellular tissue is pressed in by the external atmospheric pressure, which can be felt from without. This tissue in every wound is infiltrated with seroplastic effusion, which becomes highly vascular; in a similar manner the cellular tissue at the end of the tendon is metamorphosed. The inflammatory new growths are developed in the tissue surrounding the tendon in a similar manner in which the fragments of bone are surrounded by callus. It fills up the space between the divided tendon; an internal callus cannot develop in tendons because there is no medullary canal. The drawing shows the process in this stage about the fourth day (Fig. 122). This temporary tissue soon becomes permanent, the new inflammatory

FIG. 122.



Diagrammatic drawing of a tendon four days after it has been subcutaneously divided.

growth is metamorphosed into connective tissue; at the same time new growth has taken place at the ends of the tendon, which has merged into the tissue between them. The whole of the new tissue between the ends of the tendon gradually contracts and takes on the character of a tendon; in this way the tendon is completely regenerated. The order of events just described does not always progress so quickly, but, as in fractures, an extravasation of blood may be present between the ends of the tendon. This is surrounded by inflammatory new growths, and becomes partly organised; it takes some time, however, before it is completely absorbed and the regeneration of the tendon completed. Prolonged extravasations of blood materially retard the reparative process, in that they not only, by means of their size and the long time required for their

absorption, retard the reparative process, but that they give rise to suppuration and ichorous discharge. The operation and the reparative process in myotomy are similar to what has just been described. You have heard that the tendons are perfectly repaired, and that the effused matter between the cut ends contracts and shortens, and you will properly ask why, if this be the case, the operation is undertaken, seeing that the tendons are but very slightly lengthened by it. My answer is that tenotomy in itself is of little or no use in remedying the contraction, but that cicatrised tendon is much easier to stretch than the tendon of a contracted muscle or the latter itself. Tenotomy is only of value on account of the after-treatment, and materially advances the cure when the contracted muscles, fasciæ, withstand all attempts at stretching. The new tissue must not be allowed to contract completely, but must be stretched while still fresh; the orthopædic treatment of a club-foot may be commenced as soon as ten or twelve days after the division of the tendon, whether it consists of extension by apparatus and manipulation or plaster-of-Paris bandages.

The most favorable results are at first possible after tenotomy, the wound quickly heals and an elastic tendon is formed. When, however, there is a prolonged suppuration, the skin is implicated, and the cicatrix cannot be stretched for six or eight weeks without lacerating the skin and causing fresh suppuration. It is obvious that slight degrees of club-foot can be successfully treated without tenotomy; on the other hand, tenotomy is of great value in effecting a cure in the more advanced stages. From what has been said you can easily see that the indications for tenotomy and orthopædic treatment often coincide. This does not always happen; in some cases tenotomy has but little scope, in others it is of great value. It is clear enough that we can divide subcutaneously any contracted tendon; it is another question whether such a proceeding is advisable. It is impossible for us to discuss every possible case, but I will mention those tendons which are most frequently divided. In the neck the two portions of the sterno-cleido-mastoid muscle at their attachments to the clavicle and sternum; in the arm tenotomy is seldom performed; I advise you strongly against tenotomy in the fingers or toes; all tendons which are encapsuled by distinct sheaths are contraindicated for tenotomy; the reparative process in these cases for obvious reasons cannot progress as it does in tendons which are only surrounded by loose cellular tissue; as a

rule suppuration with serious consequence sets in, or the cut ends remain ununited. In the thigh the contracted adductor muscle, after coxitis, may be divided if it is impossible to overcome its contraction under chloroform. The same holds good with regard to the biceps femoris, semi-tendinosus and semi-membranosus, which may be divided close to their attachments to the fibia and fibula. In the foot the tendo Achillis is most frequently divided, and later on the tibialis anticus, tibialis postius, and the peronæi. In the stretching of ankyloses the operation of tenotomy was formerly much resorted to, it is now an unnecessary proceeding. For example, when in a case of ankylosis of the knee the muscles are not united to a cicatrix, they can gradually be extended under chloroform, that is to say, when the contraction is caused by muscles and not connective tissue bands, which is seldom the case. With reference to contracted muscles of the eye such as strabismus, I have nothing to say; it is treated of under the heading of ophthalmalogy. Occasionally it is necessary to divide contracted tendons through paralysis with the object of lessening the contraction, and later of extending the contracted muscles, and giving more room for the play of the opposed muscles.

The antagonistic pressure is taken off and the equilibrium thus restored. In complete paralysis the only object of tenotomy is to enable the foot to allow of the adjustment of some instrument which can bear the superincumbent weight of the body.

With regard to subcutaneous division of fasciæ there is not much to be said. The division of the band of fascia lata which forms in flexed positions of the thigh is frequently resorted to, especially as it does not allow of any appreciable extension. The plantar fascia when sketched is also divided with good results.

Where division of fascia is most desirable, that is, in contractions of the palmar fascia, we are left in the lurch. The cause of this contraction, which we have not hitherto noticed, is not clearly understood. Through the shrinking of this fascia first one, then all the fingers are curved into the palm of the hand, which is thus rendered almost useless. I once allowed myself to be tempted to divide this fascia by the description given by Dupuytren of the results which could be obtained. Such profuse suppuration supervened that I was extremely delighted when it at last ceased. The hand remained closed just as it was notwithstanding all orthopædic treatment. Slight ameliorations quickly vanished, and I regard

this deformity, when much advanced, as incurable. Some time ago Barbieri obtained a very favorable result in a not very advanced case by means of gradual extension and friction, but this treatment is an affair of many years if any permanent result is to be hoped for.

Division of ligaments is not often necessary; although I have frequently cut through the small ligaments of the tarsal bones when they were stretched, and notwithstanding this I have in this way frequently opened the small joints subcutaneously, no outward results followed. B. v. Langenbeck has divided the external lateral ligament in genu valgum, and thus opened the joint. This operation has only been resorted to in extreme degrees of deformity, but the result has been sometimes most successful. I had not seen it before this, and had not ventured on it myself for fear of suppuration of the joint. A few years ago I performed this operation in an extreme case of genu valgum in a young man. The wounds quickly healed without any suppuration, and the results of the orthopædic treatment were most extraordinary, a perfect cure resulted. The patient left the hospital with perfectly straight legs.

Altogether I find the operation is seldom required; I have lately always broken down these ligaments in genu valgum under chloroform. In other cases I have performed osteotomy on the upper part of the tibia and overcome the distortion by bending down the tibia.

We have to consider the division of contracted cicatrices, and the extension of new cicatricial growth. Would it not be much better not to allow of the formation of cicatricial contraction? Would it not be preferable during the healing of a large wound, as of the elbow-joint, to keep the arm extended and thus eliminate the formation of a cicatrix? The idea is certainly a good one, but the results are seldom successful. In the first place such wounds in which contraction is abolished heal very slowly, and when at last they are healed the contraction results all the same. I remember very well such a wound in a child who had burnt his elbow, whom I had to dress every day when assistant in the Berlin clinique. The arm was kept extended on a splint and the healing lasted six months. At last the child was sent out with a movable arm, and I was very proud of the lengthened cure. Two months afterwards I saw the child again with a completely contracted cicatrix. The arm was fixed at an acute angle and immovable. Later I lost sight

of the little patient and do not know what became of him. One thing was perfectly clear to me, I had tormented the child for months in vain. For my part I have been cured of that idea by several similar cases; much can be done by orthopædic treatment during the cicatrising of the wound. I advise you, first of all, to let the wound heal up completely, as it will; the extensive wounds which are seen in children will give you enough to do to cure; they always heal slowly and easily take on a suppurative form. In the course of some months, it may be years, the cicatrix, as the vascularity, disappears and a cutis and cellular tissue are developed, loses its toughness, becomes softer, more easily stretched and elastic. It follows, then, that the cicatrix ameliorates in time when once mobility has been set up. The mode in which the melting down of the cicatrix may be hastened by compression and extension has already been mentioned. When at last the cicatrix has shrunken to its utmost, you may excise it completely or piecemeal with advantage, so that after each excision it heals by first intention and in place of a thick inelastic scar-like band, a fine, linear cicatrix is left which is much more readily stretched than the original cicatrix. If suppuration sets in or the edges of the wound gape widely after these operations, then, as in similar cases of tenotomy, the result is dubious. There arises a broad granulating sore which gradually cicatrises, and is as wide, long, and firm as the original scar. Excision of the cicatrix may also be successfully practised in completely contracted, string-like, thin cicatrices. In those cases of large, firm cicatrices such as are met with in burns of the neck, excision alone is not sufficient. We must endeavour to cultivate a new skin in the place of the old. This may be done by twisting round a piece of the adjoining skin or by transplantation of a piece of skin in the way usually adopted in plastic operations and which I cannot here enter upon. Reverdin's experiments in transplanting skin in order to restore destroyed tissue showed that the wounds healed rapidly, but that after from two to three weeks the transplanted pieces of skin vanished without any perceptible reason, and that the wounds returned to their original condition. There are, however, some cases of ectropion which have been permanently cured by these means.

The treatment of these deformities which result from paralysis, in which the paralysis is sometimes complete, sometimes partial, has yet to be considered. I have already told you that tenotomy

is also resorted to in these cases, although it is only a secondary part of the treatment. The treatment of these conditions must be directed in a great degree to the paralysis itself. The cure of the deformities will depend upon the cure of the paralyses. This opens up the vast field of neurotic pathology, which you will be able to study better in the lectures on medicine and clinical medicine. There is a large class of cases in which *à priori* you will resort to all the treatments for paralysis: in tumours of the brain, apoplexy, chronic encephalitis, traumatic disease of the spinal cord, lacerations of nerves, &c., therapeutics are apparently powerless. Other cases of disease of the spinal cord in children, with paresis of the lower extremities, offer sometimes, a relatively bad prognosis.

The treatment by cod-liver oil and iron, as well as baths with malt and salt in them, will be advantageous. Time, however, is an important factor in the repair of lesions of the spinal cord, about which, unfortunately, we know but little. The muscles may regain their sensibility spontaneously. Those cases in which there is no complete paralysis or paraplegia, but only a paresis of several groups of muscles, allow of a favorable prognosis. In such cases there are two chief modes of treatment—gymnastics, electricity. The use of gymnastics arouses the dormant, ill-developed contractility of the muscles, and calls them into play. Definite movements at definite times are ordered; this has lately been called the "Swedish gymnastic cure," which consists in ordering the patient to perform computed exercises with certain muscles, a certain amount of opposing force being applied to the patient's movements. For example, I hold your arm firmly extended; you then bend it, while I oppose you by a slight counter pressure. In each individual case the appropriate treatment must be sought out. This form of gymnastics has lately been much advocated, and has answered well; it is, however, obvious that no gymnastics can be of any service in complete paralysis.

The second means which we have at our command is electricity. In the application of this remedy great advances have lately been made. The apparatus used for this purpose is very simple, easily carried about, and so constructed that the operator can increase or diminish the current at pleasure. The mode of applying the electricity has been much improved; formerly it was the custom to electrify first one muscle, then others of a limb, the pole being shifted irregularly; now we can galvanise an isolated muscle. The French physician,

Duchenne, of Boulogne, has made some meritorious observations on this point. The spots on which to place the rheophores in order to contract this or that muscle were found out quite empirically by Duchenne. Later, Remak recognised the fact that, as a rule, they should be applied to those spots at which the largest branches of motor nerves enter the muscles. Still more recently Ziemssen has most carefully worked at electro-therapeutics, and his book is an evidence of thorough practical knowledge of the subject as well as of trustworthiness. The treatment is as follows: One or two sittings are held daily at which first this, then the muscle is methodically galvanised. The sitting may last half or three quarters of an hour, but not too long, lest the feeble nerve force might be altogether destroyed by excessive irritation. Much harm can be done by the immoderate use of galvanism; the physician must always superintend the treatment and regulate the strength of the current to be applied. It is usually easy enough to see which muscles are incapable of excitation by the galvanic current. However, we must not despair when no effect is produced at the first sitting; sometimes after several sittings the effect is produced.

A very ingenious method of removing contractions has lately been used with success by Barwell—continued traction in the direction of the weakened muscles. For example, strong elastic bands are applied to the outer border of the foot and the inner side of the tibia close to the knee-joint, which in this way keep up a continued contraction as artificial muscles. It seems to me to be a very rational mode of treatment, and should certainly be widely tried. I myself have resorted to this mode of treatment in several cases with most extraordinary results; Lücke and Volkmann also speak highly of it. In paresis, movement of a few muscles may suffice for walking, if the whole of the leg is supported by a splint apparatus, and thus acquires sufficient steadiness, which it would not have from the muscles alone. Such apparatus applied to the lower extremities need not by any means be regarded as an *ultimum refugium*, only they assist the cure in so far as they allow of the patient's walking about without help, except from a walking-stick. The movements of walking which are thus brought to bear on the semi-paralysed muscles act gymnastically; the patient, when supported in this way, brings into use those muscles which retain their functions, while, if he continually lies down or sits, they remain inactive and gradually atrophy. The apparatus is also of value in

keeping the leg extended and the foot in its normal position, and thus retarding the progress of contraction.

Gymnastics, electricity, artificial muscles and splint apparatus, combined with internal remedies, occasionally with appropriate hydropathic treatment, often give very favorable results. And, although many of the cases are incurable, on the other hand there are not a few which can be considerably benefited.

LECTURE XLII.

CHAPTER XX.

ON VARICES AND ANEURISMS.

Varices: Varieties. Etiology. Various seats. Diagnosis. Phlebolithes. Varicose ulcers. Therapeutics. Varicose lymphatics. Lymphorrhœa. Aneurisms: Inflammatory processes in arteries. Aneurisma cirsoidea. Atheroma. Varieties of aneurism. Later changes. Appearances. Results. Etiology. Diagnosis. Therapeutics: Compression. Ligature. Injection of Liq. Ferri. Extirpation.

By varices are understood dilatations of veins; there are various forms, and the diameter is usually involved as well as the length of the vessels. Lengthening can only happen when the vessel is twisted and takes an erratic course, as in inflammation of the smaller vessels. In many cases the lengthening is not marked, and the diameter of the vessel is unequal; moreover, the vessel is fusiformly or saccularly dilated at different spots, especially opposite valves.

FIG. 123.

Varices in the course of the vena saphena.

Most commonly the larger veins of the subcutaneous tissue are implicated. Sometimes the deep veins in the muscles are involved,

and sometimes both sets together. A varicose condition may, however, arise in the smallest veins of the skin itself, which are hardly visible to the naked eye. When this is the case the skin presents an even bright blue colour, with a rough appearance. As a result of these dilatations of the veins, which develop very gradually, more serum than is usual is poured out from the capillary vessels, while, on account of the increased pressure on the walls of the vein. and the consequent incompetence of the valves, the excentric pressure in the capillaries is considerably increased. The thinning of the walls of the vessels and the transuded nutritive material gives rise gradually to hypertrophy of the surrounding tissue. There is first a serous, then a cellular, infiltration and thickening of the surrounding cellular tissue. Red blood-corpuscles may transude through the walls of the capillary vessels. The pressure in standing and walking of the distended veins upon the surrounding structures finally gives rise to inflammatory irritation. We have already mentioned, that as this process goes on the structures become changed, and chronic inflammation and ulceration occur. In this way, not only ulcers, but many other forms of inflammation of the skin of a chronic nature, as eczema.

We must now consider the causes of varicose veins. It is *à priori* likely that they are caused by some impediment to the flow of the blood back through the veins. Pressure, or compression of the veins, or an alteration in the diameter of the veins in some way. The impediment is not a sudden one, for sudden obstruction to the venous flow only gives rise to œdema. The ligature of a large vein does the same thing, and develops a thrombosis in addition. The pressure must act gradually on the venous trunk. This alone is not sufficient, for considerable pressure frequently gives rise to no varicosity, but causes the growth of collateral vessels, and thus only slight œdema or no symptoms whatever arise. A disposition to dilatation of the vessels must be present at the same time, with a looseness, elasticity of the walls, and, perhaps, an irritable condition. The minute investigations of Soboroff show that there are changes in the walls of the veins themselves. Soboroff examined the vena saphena and its branches. He found that these veins varied in different people in the normal condition as regards their different layers, and that sometimes adjacent spots of these veins were variously affected. This is extremely interesting, because it explains why the appearance of the varicose condition in similar

cases is yet so varied, and depends entirely on the individual condition. We can easily distinguish between thickened or attenuated walls. Usually the muscular fibres are hypertrophied and the endothelium intact. The difference of diameter of the walls of the veins is the chief cause next to the thickening of the adventitia. The connective tissue which encloses the muscular fibres causes a slight degree of thickening of the intima, not seldom a sclerosis of this coat is found—as in arterial sclerosis. The anatomical conditions in dilatation of the veins resemble those which are present in the urinary bladder and heart under the same conditions. The muscular fibres seem to hypertrophy from increased function, increased nutrition is aided by the vasa vasorum, so that the connective tissue, especially the adventitia, is markedly increased. When this increased nutrition of the walls of the vessels ceases, atrophy and flaccidity supervene.

A tendency to varicose veins is in many cases hereditary; diseases of the blood-vessels especially are frequently hereditary, diseases of the arteries, as well as if the veins and capillaries through whose abnormal distension nævi or so-called mothers' marks are caused. These are well known by the laity to be hereditary. Varicose veins are more common in women than in men; this is to be attributed to repeated pregnancies: the gradually enlarging uterus presses on the venæ iliacæ communes and later still on the vena cava, and in this way œdema is caused in the feet as a result of pressure on these veins. Varicose veins often arise throughout the whole distribution of the vena saphena, also occasionally in the course of the venæ pudendales and labia majora. The origin of varicose veins is not so easy to discover in men. Large accumulations of fæces pressing on the hæmorrhoidal veins may give rise to varices, although this is not often diagnosticated. You will see many men with disproportionately long legs, and this condition may in some cases undoubtedly favour stagnation in the veins. Moreover, it is possible that large masses of fat in obese persons, or contractions of the processus falciformis of the fascia lata, may give rise to retardation of the blood-current in the vena saphena, and later on in the vena femoralis. Anatomical conditions, as far as I know, do not exercise any influence in this way. The impediment to the circulation would not seem to lie altogether in the course of the larger veins; it seems possible that gradual narrowing and obliteration of the vena femoralis below the junction of the vena saphena

may give rise to the establishment of a large network of collateral vessels. Varicose veins may develop in many other parts of the body, especially in the rectum and spermatic cord. Varices of the vena hæmorrhoidales in the lower part of the rectum, called hæmorrhoids (from αἷμα, blood, ῥέω, to flow), are especially common in persons who lead a sedentary life.

Varicose veins in other parts of the body are very rare; they may appear in the head without any obvious cause; they may result from injury, causing an intercommunication between an artery and a vein, and are then called varix aneurismaticus, which has been spoken of in the second chapter. In the atlas of pathological anatomy of Cruveilhier you will find a rare drawing of an extensive varicose condition of the abdominal veins; there is a similar preparation in the museum of pathological anatomy at Vienna. Quite recently a similar case came into my clinique; a large substernal strumatous swelling had pressed on the right innominate vein. When the veins of the skin are involved, the diagnosis of varices is not difficult; when the deep veins among the muscles are implicated, the diagnosis is uncertain. In the thigh and leg the twisted meandering veins are often so plainly felt under the skin as to be easily recognised as such. In other cases only a few bluish, fluctuating aggregated knots can be seen, and that chiefly at the spots where the veins dilate, and near valves. Occasionally we find here hard, firm, roundish bodies; vein stones, phlebolithes (φλέψ, vein, λίθος, stone); on examination they appear to be small clots, which were originally fibrinous, and aftewards calcareous and resembling small peas. Varices of the lower extremities in the large majority of cases give no great trouble beyond perhaps a feeling of distension and weight in the leg after a long walk or continued standing. In both cases thrombosis may arise in one or more cf the dilated veins; inflammation of the vein and the surrounding cellular tissue results, and, even when the inflammatory process has been partially subdued by timely treatment, ulceration or abscess may eventually set in. The treatment is the same as that which we have recommended in traumatic thrombosis and phlebitis. One other dangerous complication may arise—rupture of the varicose veins, but this is rare. The bleeding is easily controlled by compression, and is only dangerous in the absence of medical assistance. A varicose ulcer may arise as a consequence of ruptured varix, but this is seldom the case. It may also happen

that the small opening of a ruptured varix may imperfectly close, and after a little while break open again, giving rise to bleeding, close again, and so on for some time. This condition is called a varicose fistula; it may be cured by rest and pressure, or the varix may be extirpated. When the whole skin and subcutaneous cellular tissue of a leg is much indurated, and the thickening has involved the adventitia of the superficial veins, the latter are embedded in firm rigid skin and feel like canals or gutters. I call your attention particularly to this because you might otherwise, in such cases of induration of the skin, easily overlook the varicosities entirely.

In the *treatment of varices* we may at once declare ourselves so far helpless that we know of no remedies by means of which we can obviate the disposition to these affections of the veins. In most cases we cannot even prevent the pressure from which they result, and must, therefore, come to the conclusion that they are for the most part incurable, *i.e.* that we have no means of reducing the morbidly dilated veins to their normal calibre. We must admit, for many cases, that the formation of the varices, regarded in a physiological sense, is a natural compensation for abnormal pressure in the vascular system, and that we can have no prospect of curing them so long as we are unable to remove their causes, for even if we removed one or more of these diseased veins, fresh channels would soon become developed in their stead. On this account alone I avoid all operations the object of which is to excise one or more varicose knots from the leg. If you bear in mind that individual varices in themselves cause little or no inconvenience, and that every operation upon the veins may become dangerous to life by complication with thromboses and emboli, you will agree with me when I declare the operation for varices utterly uncalled for. Nevertheless, these operations are very often performed in France, and not unfrequently with a fatal result. There are a great many ways of operating, but I shall not say much about them. The most ancient method, which was adopted already by the Greeks, consists in laying free the varicose veins, and then either cutting or tearing them out. Later on, the ferrum candens was much used and coagulation of the blood in the veins brought about which led to a partial or total obliteration of those vessels. A solution of perchloride of iron may also be injected into them with a very fine syringe, which, as you are aware, rapidly causes coagulation of the blood. Lastly,

ligatures may be applied to the veins, especially subcutaneous ligatures on Ricord's plan, or subcutaneous rolling (enroulement, Vidal), slight operative manœuvres which I will show you in the operative course—very ingenious methods but unfortunately of no use and not always free from danger.

Are we, then, not to attempt anything against varices? Yes; we must endeavour to keep them within certain bounds, and thereby prevent or reduce to a minimum their evil consequences. There is one means for doing this, namely, *continued pressure,* which must only be carried out, however, to such an extent as to be easily borne by the patient. We emply two different kinds of mechanical contrivance for causing pressure in these cases—laced stockings and skilful bandaging. The laced stocking consists of a leather stocking of uniform thickness cut open on one side and furnished, like stays, with an apparatus by means of which it can be drawn sufficiently tight; or an elastic material of cotton or silk spun over india-rubber threads may be used, which is known to you already since most braces are made of it. These stockings, which must be applied very carefully and worn constantly, are, unfortunately, rather expensive, and must be replaced frequently, since they cannot be washed, so that they are, in reality, practically useful for well-to-do people only. For most cases skilful bandaging suffices. For this purpose you should use a bandage from two to three fingers' breadth and apply it from the toes to the whole leg as far as the knee, but leaving the heel free. Such a bandage may be worn five to six weeks, and prevent the formation of ulcers even when there is considerable infiltration of the skin, since it checks at the same time the further development of the varices.

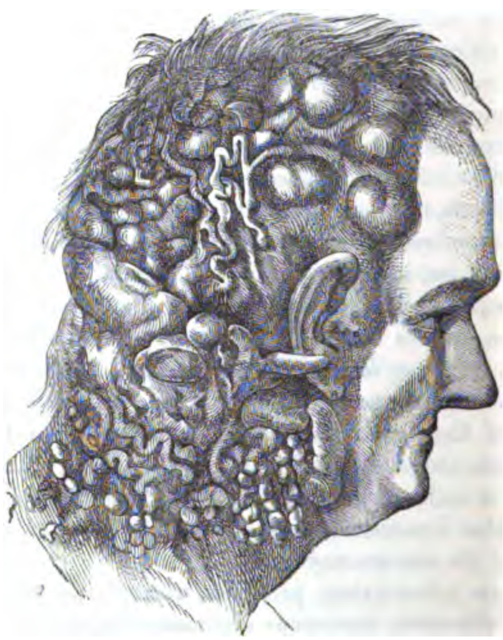
In very rare cases, *varicosities* of the *subcutaneous lymphatic vessels* of the extremities occur. The inner side of the thigh at its upper third is the favorite seat of this disease, which must have already attained a very high degree to become distinctly visible. In most of the cases on record, convolutions of lengthened lymphatic vessels arise which sometimes become confluent and form cavities. Perforation of the skin then not unfrequently takes place, and a fistula is formed, *i.e.* a large quantity of lymph is discharged daily, which is generally entirely serous, but in some cases had a milky appearance. A cure by pressure can seldom be effected, and it has generally become necessary eventually to extirpate the whole convolution of lengthened lymphatic vessels.

It is now some time since anything has been said concerning *traumatic aneurism;* you will remember, however, that I alluded thereto when speaking of wounds made with a sharp-pointed instrument, and that I told you then that an aneurism is a cavity or sac which is in direct or indirect communication with the canal of an artery. That such cavities may develop themselves after injuries to arteries by thrusts, or after subcutaneous lacerations or contusions, you are already aware. We have not now, however, to speak of these traumatic, so-called false aneurisms, but of the *true aneurism,* which develops itself gradually in consequence of disease of the wall of the artery. To represent to you clearly how this is brought about, it will be best for us to start from the anatomical conditions. You do not know much as yet concerning diseases of the arteries; with the exception of the formation of thrombi after injuries, the development of the collateral circulation, and the atheromatous process briefly mentioned when speaking of senile gangrene, no other affections of the arteries have been described. With these affections, moreover, the subject is almost exhausted, except that the consequences of atheromatous disease have, as yet, been treated of in a very one-sided manner only. Of the different parts of which an artery is composed it is the muscular and the inner coat which most frequently become diseased, and especially, as it appears, diseased primarily. The middle coat is composed of muscular cells and some connective tissue, the inner coat consists of a vascular elastic lamella, fenestrated membranes, and the very thin endothelial coat. After injuries to arteries, it is very easy to recognise that the wall of the vessel swells and continues thickened; the plastic infiltration of the walls of the arteries may also lead to suppuration, so that small suppuration-nests may form in them—a process much less frequently observed in them, however, than in veins. In these processes a relaxation of the membranes occurs, the inner coat is more easily separable from the middle coat than usual, the latter becomes softer, the muscular cells may become partly broken up, and, in consequence of this diminished resistance of the walls of the vessels, dilatation of the arteries may ensue.

Such acute inflammatory processes, with plastic new formation and partial dilatation, may occur spontaneously, and if we do not possess any special observations on the subject, there can be no doubt, from analogy to other tissues, that a spontaneous, idiopathic, acute and subacute inflammation of the arteries may run this course,

and probably occurs simultaneously with acute processes of inflammation in other tissues. At all events, these acute spontaneous inflammations of the arteries are extremely rare, much more so than the chronic cases. Only one form of aneurism depends, perhaps, upon such a subacute diffused process of inflammation of the arteries with decreased resistance of their walls, namely, the *aneurysma cirsoideum*, or *aneurysma per anastomosin*, called also *angioma arteriosum racemosum*. This kind of dilatation of arteries is entirely different from the aneurisms to be mentioned later on; it is a question here, not of the circumscribed dilatation of a part of an artery, but of the dilatation of a great number of arteries lying close to each other, which are moreover much convoluted—a sign that the arteries have become considerably increased in length. The *aneurysma cirsoideum is, therefore, a convolution of dilated and lengthened arteries*. To render this change possible, a considerable new forma-

FIG. 127.



Aneurysma cirsoideum of the scalp in an old woman; a small tumour was said to have existed at birth and to have increased gradually to this extent. According to Breschet.

tion must take place in the walls of the vessel in the long direction also. The dilatation results perhaps from atrophy of the muscular coat.

It is usually assumed, without any proof however, that aneurisms of this kind result from paralysis of the walls of the arteries; but even if such paralysis might account for a moderate degree of dilatation of the vessels, the cause of the paralysis itself remaining quite unexplained, (in complete paralysis of the lower extremities, for instance, no dilatation of the arteries takes place), the increase in length of the arterial tube, which can only result from a new formation of wall-elements, is not rendered more intelligible thereby. I believe, as already remarked, that this kind of dilatation of the arteries, which very closely resembles the inflammatory dilatation and convolution of the vessels, must be referred to a process of inflammation in the arteries, and *not, in fact*, to the chronic form of inflammation with formation of atheroma, to be described later on, but to a more subacute diffused inflammation with predominant new formation of tissues. Several ætiological facts also tend to support this view. These aneurisms may not unfrequently be shown to occur after blows, thrusts, &c., and are observed most commonly at points at which several small arteries anastomose with each other, especially on the occiput, temples, and vertex. We might regard an aneurism of this nature as an excessive development of the collateral circulation. The dilating collateral arteries also become, in addition to the dilatation, much convoluted, and the process which leads to the dilatation and convolution of the vessels is evidently in both cases the same. It must be mentioned, further, that aneurisms of this kind become developed especially in young subjects, in whom chronic affections of the arteries leading to other forms of aneurism are rare. The diagnosis of aneurysma cirsoideum is very simple if, as is usually the case, it lies beneath the skin; aneurisms of this kind have, indeed, been met with much more deeply seated, *e.g.* about the glutæal artery, but they most frequently occur upon the head; we here feel the pulsating convoluted arteries plainly, and can see them pulsating, so that the disease is easy to recognise; it is rare, on the whole.

It still remains to be mentioned here that the walls of the arteries may become diseased in an acute or chronic form by the spreading of a process of ulceration or suppuration from their neighbourhood, first to their outer coat and then to the other coats, so that the

latter become implicated; this is by no means common with acute abscesses, but more frequent with chronic processes of ulceration. To give you an instance of this, it not infrequently happens during the formation of cavities in the lungs that the ulcerative process involves the walls of the smaller arteries, and that the outer coat becomes partly softened and destroyed, or that the softening results from the formation of tubercle in the walls of the artery. The consequence of this is that the artery becomes dilated at that point and a small aneurism formed, the bursting of which occasions profuse pulmonary hæmorrhages. Other processes of ulceration also may, although this is rare on the whole, make their way to an artery and destroy its coats, so as to cause the artery to burst and, if it be a large branch, produce fatal hæmorrhage. I have known several such cases; in one, a deep-seated abscess, in the neck of an old man had burst into the pharynx; this was diagnosed from the gradual formation of a painful swelling in the neck and subsequent copious expectoration of stinking pus; the patient had not been many hours in the hospital before he brought up suddenly an enormous quantity of blood, rapidly became asphyxiated, and died; the post-mortem examination showed that, in consequence of suppuration of the cellular tissue around the art. thyroidea superior, that artery had poured out a large quantity of blood, which had passed directly into the larynx and caused death from suffocation. In another case, repeated arterial hæmorrhages from the right ear occurred in a young man who had caries of the petrous portion of the temporal bone; I diagnosed an abscess in the lower part of that bone, with suppuration of the internal carotid artery. The hæmorrhages could not be controlled by plugging the ear, and I tied the right common carotid artery. The hæmorrhages ceased for about ten days and then recommenced. Renewed plugging of the ear, followed by digital compression of the left carotid artery, having had no permanent good effect, I tied the left common carotid also; two days afterwards the patient died, however, from profuse hæmorrhage from the right ear, nose, and mouth; the abscess, which was filled with blood, and might now be regarded as a false aneurism, had also burst into the pharynx. The post-mortem examination entirely confirmed the diagnosis.

We now come to *true aneurisms*. At an advanced age we very frequently find the arteries strikingly thick and hard, sometimes also convoluted, especially arteries of the diameter of the radial and

smaller ones. If we examine such rigid arteries more closely, we find the inner coat thickened, of cartilaginous hardness, the canal of the vessel more rigid than usual and gaping; here and there the artery is even of a chalky hardness, thoroughly calcified, or ossified. These chalky portions are not situated diffusedly in uncertain parts of the wall of the vessel, but are in the form of circles corresponding to the transverse, muscular layers of the middle coat; it is the muscular fibres of the vessel which have become chalky. In individuals with such arteries, we find in the aorta, and in the larger branches given off from it, whitish-yellow spots on the inner surface, streaks, bare-looking places, &c., partly chalky, partly rough as if eroded, with undermined edges. If we make incisions through these points, the whole of the inner coat is seen to be of cartilaginous hardness, yellowish white, or completely chalky and of bony hardness, or crumbling. When this disease has attained a high degree, the arteries are dilated at certain points so as to form pouches. These are the features of atheroma of the arteries as we meet with it in the dead body. We observe in it different stages, either contiguously or in different arteries.

If we examine these points more closely with the microscope, especially in fine transverse sections through the portions presenting the different appearances, the more minute process is seen to be as follows: the first changes take place in the outer layers of the inner coat, especially at points nearest to the middle coat; a moderate accumulation of cells occurs here. The young cells may lead to formation of connective tissue and callous thickening of the arterial wall, but these last, for the most part, but a very short time; while fresh cells are forming at the periphery of the disease-nest, the first break up into a granular detritus, a pulp consisting of fine molecules and fat, which remains, as in the process of fatty degeneration, rather dry; the destruction thus goes on slowly on the surface, the nutrition of the middle coat suffers as well as that of the innermost layers of the inner coat; the muscular cells of the former undergo granular and fatty decay, as do also the elastic lamellæ of the inner coat; this process advances inwards to perforation of the last layers and epithelial coat and the cavity filled with atheromatous pulp opens into the canal of the artery. The atheromatous process, commencing as a cavernous ulcer, has led to an open ulcer with undermined edges; you see that it is the same mechanism with which you are already familiar in the skin and

lymphatic glands; it is a chronic inflammation terminating in caseous degeneration, or, as this pulp is called here, in the formation of atheroma.

I have given you herewith the most essential part of the process, so far as it is of interest for the formation of aneurisms; it presents, however, many variations, and is modified essentially in its course by the differences in the construction of the arteries. The less the inner coat is developed so much the less atheromatous pulp becomes formed, for the latter originates chiefly in the decay of that coat. Let us first of all take into consideration the small arteries, disease in which we can best study in the minute arteries of the brain. We there find the accumulations of cells chiefly in the outer coat, which is less and only secondarily affected in the larger branches in this disease. Almost the whole outer coat breaks up into cells, the few muscular cells become atrophied, the fine vitreous membrane which serves as an inner coat is extremely elastic, and the softening of the outer coat caused by the cellular infiltration soon leads to dilatation, and eventually to bursting of the artery, because its walls are no longer strong enough to resist the pressure of the blood. A plastic formation also sometimes takes place in the outer coat; knotty vegetations become formed which consist partly of newly-formed fibrous, partly of homogeneous connective tissue. We cannot follow out this question further here, especially as it is of little importance in a surgical point of view. Chalky and fatty degeneration of the muscular coat also occurs in the small arteries of the brain, along with the plastic infiltrations of the outer coat, but is not very common. Let us now go on to arteries of the diameter of the basilar or radial, &c. The plastic process in the outer coat here sometimes competes successfully with that in the other two coats, although pulpy decay and chalky degeneration of the latter occur. At one time thickening and convolution of these arteries predominate, at another rather decay and softening, with consequent dilatation and aneurismal formation, for if the middle and inner coats are softened at some point to form an atheromatous pulp, the outer coat is then no longer strong enough to resist the pressure of the blood, and a pouch is formed.

If we now take into consideration, lastly, the large arterial trunks, the aorta, the carotid, subclavian, iliac, and femoral arteries, you know that in them the muscular coat is reduced to a minimum, or is even altogether wanting in places, while the inner coat, on the

contrary, consists of a great number of elastic lamellæ, and impinges almost immediately upon the outer coat more or less copiously supplied with elastic fibres. The plastic process in the outer coat is here least active; the pathological change, the disturbance of function, shows itself predominantly in rapid decay or chalky degeneration of the pathological new formation, which takes place partly at the boundary of the inner coat, partly in that coat itself. There are also cases, no doubt, in which more extensive circumscribed new formations of connective tissue, in the form of cartilaginous wheals, take place in the inner coat, as has been mentioned already, but this occurs more rarely than the metamorphosis to an atheromatous pulp. The latter becomes developed most frequently in the last-named large arteries, and it is in them, therefore, that aneurisms form predominantly.

If you examine this fully developed atheromatous pulp with the microscope, you find in it, in addition to the already mentioned molecular and fatty granules, fat crystals, especially cholesterine in a crystalline form, further, crumbs of carbonate of lime and perhaps crystals of hæmatoidine, resulting from the formation of coagula of blood, at the rough points of the arteries, from the colouring matter of which the hæmatoidine becomes developed.

You have now a general idea of the atheromatous process in arteries of different calibre, and understand how it may lead to partial dilatation of the canal of the artery, and formation of aneurism by softening of the walls of the vessels. The form of this dilatation may vary somewhat according as the artery is uniformly or irregularly diseased in its whole periphery, and according as softening or chalky formation predominates.

The dilatation of the artery may be perfectly uniform in a certain length; it is then called *aneurysma cylindriforme;* if the dilatation is more spindle-shaped, *aneurysma fusiforme*. If the softening of the artery is confined to one side of the vessel, a pouch-like dilatation occurs, the *aneurysma saccatum*, which may communicate with the canal of the artery by a larger or smaller opening. A further difference in the form of the aneurism may consist therein that either all the coats participate uniformly in its production, or that the inner and middle coats are completely softened and destroyed, and only the gradually thickening outer coat and infiltrated surrounding tissues form the sac. Lastly, the blood may, under the circumstances just mentioned, make its way between the middle

and outer coat, and detach the two coats from each other, as in an anatomical preparation of the layers of the artery; it is then called *aneurysma dissecans*. These distinctions might be carried still further, but they have very little practical value. I will only add here that with the subcutaneous bursting of an aneurism, formed by all the coats of an artery, the aneurism assumes rather the anatomical characteristics of a traumatic or false aneurism. I saw a case a short time ago in which, in an apparently healthy man of fifty, an enormous tumour suddenly appeared in the thigh on his turning round in bed, which soon became recognisable easily as a diffused traumatic aneurism. I felt no doubt that the femoral artery was diseased, and had suddenly given way at a point about the middle of the thigh. After compression had been tried for a long time in vain, the femoral artery was tied, and was seen during the operation to be sprinkled with yellowish spots. The ligature held well, and came away in a month's time, but the aneurism increased in size and became painful. In the sixth week after the operation gangrene of the foot set in; I then performed amputation high up in the thigh, and the patient has recovered. A colossal false aneurism was found, and a fissure an inch long in the atheromatous femoral artery.

Of great importance is the further fate of the aneurism and its influence upon the neighbouring tissues or upon the respective extremity. As regards the anatomical changes which may take place subsequently in and about an aneurism, these consist therein that the aneurism gradually becomes larger, and not only displaces the surrounding parts, but by its pressure and pulsation causes them to waste. This holds good not only for the soft parts, but also for the bones, which gradually become broken through. This occurs especially with aneurisms of the aorta and anonyma, which may cause wasting, partly of the bodies of the vertebræ, partly of the sternum and ribs, in the form of lacunar corrosion, as in caries. Inflammatory processes may also set in in the immediate neighbourhood of aneurisms, which seldom lead to suppuration, however, often become chronic, and are very rarely followed by gangrene in the aneurism. Lastly, coagulations of blood very frequently occur in aneurisms; firm layers of coagulum may form on the inner surface of the sac, and these may eventually fill up the sac completely, and thue effect a spontaneous obliteration, a kind of cure of the aneurism. The worst termination is that of the bursting of

the aneurism after it has gone on increasing for a considerable time; it may burst outwards, but it much more frequently takes place inwards, especially in the case of the large arteries of the trunk, into the œsophagus, the trachea, or the thoracic or abdominal cavity. Rapid death from hæmorrhage is the natural consequence of this.

It is not our object here to inquire what may be the consequences of an aneurism in the arteries of internal organs; I will merely remark that of the coagula which form in the aneurismal dilatations, or which adhere to the rough points of atheromatous arteries, particles may become detached and carried on as emboli with the arterial current into the arteries of the periphery; these emboli will then occasionally cause gangrene. This process is not so common, however, as might be expected, because the coagula in the aneurisms adhere, for the most part, very firmly.

We must now turn our attention more closely to *aneurisms of the extremities*. They cause at the commencement slight muscular fatigue and weakness, seldom pain, in the extremity affected; as soon as inflammation sets in around the sac there is naturally pain, with great redness of the skin, œdema, and considerable disturbance of function, which may proceed so far that, with continued growth of the aneurism and continued chronic or subacute inflammation around it, the extremity may become completely useless. With the formation of extensive coagulations in the aneurism of a large arterial branch, gangrene of the whole extremity below the aneurism may ensue.

I mentioned already, when speaking of gangrene, that it may set in in consequence of atheroma of an artery as so-called spontaneous gangrene; it is there a question, however, of something different, of a diseased condition of the small arteries, the function of which becomes arrested by the destruction of their strong muscular coat, so that they cannot drive the blood on because they no longer contract. But here it is a question of obliteration of a main arterial branch by coagulations at an aneurismal point. I will relate to you a case which was observed in the surgical clinique at Zurich. A man twenty-two years old, emaciated and wretched-looking, was brought into the hospital; his left leg was of a blueish-black colour almost as high as the knee, the epidermis came off in shreds, and there was unmistakable gangrene. The examination of the arteries showed an aneurism of the left femoral artery close under Poupart's ligament, spindle-shaped and pulsating distinctly; a

second three inches lower down on the same artery pouch-shaped and firm to the touch; a third in the hollow of the knee, also firm, but not definable in shape on account of the swelling of the surrounding soft parts. Between the second and third aneurism the artery still pulsated during the first few days after the patient's admission, but decreased daily more and more in an upward direction; the gangrene showed no distinct line of demarcation, but appeared to have a tendency to spread upwards. The pulsation in the artery gradually disappeared quite up to Poupart's ligament. The patient died about a fortnight after his admission into the hospital. The *post-mortem* examination proved the existence of the aneurisms recognised during life, as well as of an extensive atheromatous condition of almost all the arteries.

If you compare this case with what I have told you of the development of the collateral circulation when large arterial branches are tied, it may appear to you that I have contradicted myself. Why does gangrene not set in if you close the artery by means of a ligature, just as it does after closure by coagula? The answer is as follows: a free collateral circulation, sufficient for the nutrition of the periphery, becomes established only in arteries which are sound and admit of dilatation; the blood passes around the ligature into the peripheric portion of the tied artery. But if a formation of coagula, having its starting-point in an aneurism, occurs in an arterial branch, the secondary arteries are generally diseased, partly calcified, or already partly closed and not dilatable. Moreover, the closure of the arterial trunk is not confined to a small point as in the case of a ligature, but extends over a very large space, perhaps even, as in the case just mentioned, to the whole artery. A circulation then naturally becomes impossible, either by means of collateral branches or in the main vessel. The arteries must be very generally diseased and the coagulation very extensive to cause gangrene, which is not, on the whole, very frequent in cases of aneurism; that would be a very sad state of things in reference to treatment, the chief object of which, as you will learn later on, is the closure of the aneurism with or without the aid of a ligature.

We now come to the *ætiology* of aneurisms. Although atheroma of the arteries is an extremely frequent disease of advanced age and occurs everywhere, the formation of aneurisms is by no means a disease of that period of life alone. In Zurich atheroma of the arteries and senile gangrene are rather common in elderly people,

but aneurisms in the extremities are rare. The occurrence of aneurisms is curiously distributed over Europe; in Germany, aneurisms of the extremities are very rare; they are somewhat more common in France and Italy, most common in England. It is difficult to give any particular reasons for this, but it is a well-established fact that diseases of the arteries in connection with rheumatism and gout occur more frequently in England than in all the other countries of Europe. As regards age, aneurisms (exclusively, of course, of traumatic aneurisms) are rare before the thirtieth year of life, more frequent between thirty and forty, most frequent after forty. Men are more liable to the formation of aneurisms than women. Special occasional causes are little known; most frequent in the extremities is popliteal aneurism. It has been sought to explain this by the superficial position of the popliteal artery, by the tension it undergoes in sudden movements of the knee, by contusions, &c. Thus this aneurism is said to be especially common in England in servants who stand behind carriages, but I must confess that this appears to me just as improbable as the usual explanation of housemaid's knee. I am inclined to think that the tendency to arterial disease, as well as gout, is based chiefly upon the hereditary character of the latter; it is also assumed that hard work and the free use of spirituous liquors predispose thereto; the last-mentioned habit is said to lead frequently to relaxation of the walls of the arteries, without atheroma, especially in England.

The diagnosis of an aneurism in an extremity is not very difficult if we examine carefully, and the aneurism is not too small. An elastic, more or less firm, circumscribed (in false aneurisms, or such as have burst, diffused) tumour is met with, which is connected with the artery. The tumour is seen and felt to pulsate, and if you apply the stethoscope to it, you hear a pulsating rushing sound, or, strictly speaking, a friction sound, produced by the rubbing of the blood against the coagula, or at the more or less narrow opening of the aneurismal sac, or by the ricochetting of the blood in the sac. The tumour ceases to pulsate if you compress the arterial trunk above it. These symptoms are, indeed, so significant that it would appear impossible to hesitate about the diagnosis, and yet it has not infrequently happened that even experienced surgeons have been mistaken at moments in which they did not think of the possibility of an aneurism, and acted hastily. An aneurism may, namely, be greatly masked if the surrounding tissues are highly

inflamed and there is much swelling of the soft parts. It may, under certain circumstances, give the impression of a simple inflammatory swelling, or of an abscess, or may even have resulted from an abscess, as mentioned already. The mistake most frequently made is that of confounding it with an abscess; we introduce a lancet and are very disagreeably surprised to see, instead of pus, a gush of arterial blood. We have no appropriate means at hand to check the profuse hæmorrhage. The situation is a most painful one, even if a self-possessed surgeon can meet the first emergency of it by compression, until he has decided what is to be done next. But I will not represent the matter to you too unfavorably, and will repeat that, with careful examination, such a mistaken diagnosis cannot easily be made. If the aneurism is very full of coagula, no pulsation may be perceptible in the tumour, or both it and the friction sound may be very slight; a further, more minute examination will enable us, however, to arrive at a correct conclusion here also. It may happen, on the other hand, that we take a tumour for an aneurism which is not one. There are certain soft tumours (mostly soft alveolar sarcoma) in the bones, especially and particularly in the pelvis, which are very richly supplied with arteries, and, therefore, pulsate distinctly. About these arteries many small aneurisms may form in consequence of softening of the mass of the tumour and of the walls of the arteries. The sum of the murmurs in these small aneurisms may amount to a distinct aneurismal murmur, and only the most exact investigation and observation can distinguish between them. These pulsating tumours in bone are regarded by many as true aneurisms; I do not believe that aneurisms arise spontaneously in bones, but that all these so-called aneurisms in bone were cases of soft sarcoma very rich in arteries. Lastly, we may also be tempted to take a tumour, which is situated immediately above an artery, and becomes raised by its pulsations for an independent pulsating tumour, for an aneurism; in short, the absence of the aneurismal murmur, the consistence of the tumour, the possibility of isolating it from the artery, and the observation of the further course of things, will save us from mistakes here also.

The *prognosis* of aneurisms differs greatly according to their situation, so that nothing can be said in a general way concerning it.

We will now speak of *treatment*, and I will merely remark provisionally that, in rare cases, the cure of an aneurism may occur

spontaneously, namely, by the complete closure of the sac, and of a part of the artery by coagula; the tumour ceases to enlarge and gradually shrinks up. It has also been observed, as already mentioned, that the inflammation around the aneurism may lead to local gangrene; if the artery was then already closed, the whole aneurism may come away in a state of gangrene without the occurrence of any hæmorrhage. These natural cures are extremely rare, but show us the way in which we may proceed therapeutically against the disease. I shall not allude further here to the medical treatment of internal aneurisms than to mention one method, namely, that of Valsalva, the object of which is to reduce the value of the blood to a minimum, and thereby diminish the action of the heart and facilitate coagulation. Repeated bloodletting, purgatives, absolute rest in a horizontal position, spare diet, digitalis internally and ice locally in the neighbourhood of the aneurism, are the remedies employed in this treatment. The results of this method are very doubtful; the patients become frightfully reduced, and the symptoms may then be less marked, but as soon as the patients are allowed to regain strength the previous state of things generally recurs. The means just mentioned may be adopted to a moderate extent for the mitigation of severe symptoms of internal aneurisms, but we shall never effect a real cure therewith; internal aneurisms must, unfortunately, almost always be regarded as incurable evils. If we turn to the surgical treatment of external aneurisms, we may start from one of two different points: we may either attempt to empty the aneurism completely, or to remove it altogether. For the generality of cases the emptying of the tumour will suffice. There are several methods for attaining this purpose.

1. *Compression.* This may be applied in various ways: (1) to the aneurism itself, (2) to the diseased arterial branch above the tumour. The latter is by far the most appropriate mode of proceeding, because even a moderate amount of pressure upon the aneurism is often painful, and may give rise to processes of inflammation in its neighbourhood. The mode of applying the pressure also varies; it may be continuous, and at the same time, complete or partial; it may be temporary, but for the time pretty complete, *i.e.* such as to check the pulsation entirely. The methods of compression are as follows: (1) compression with the finger, especially recommended by Vanzetti, and employed successfully by him and many other surgeons. It is carried out by the surgeon, the atten-

dants, or the patient himself, at intervals, for some hours at a time, until the pulsation ceases entirely; this is to be continued, if the patient can bear it, for days, weeks, or even months, until the aneurism no longer pulsates at all, and has become hard and small; (2) compression of the aneurism by forcible flexion of the limb; this method, which was first practised by Malgaigne, is especially adapted for popliteal aneurism; the leg is firmly flexed by means of a bandage, and kept in that position until the pulsation in the aneurism has ceased; (3) compression with special apparatuses, pads, &c., which must be so prepared that the pressure may act as exclusively as possible upon the artery only, so that œdema may not be caused by simultaneous compression of the veins. The pressure need not be so great as to check pulsation altogether, the object being merely to diminish the afflux of blood. Opinions as to the efficacy of compression in the treatment of aneurisms are much divided; Irish surgeons are strongly in favour of it; French and Italian surgeons have turned their attention more to this method recently, especially since the publication of Broca's excellent works, and the intermitting digital compression in particular has attained brilliant results. My own opinion is that we should first of all try compression, in the generality of cases, for the treatment of aneurism, but our present experience shows that this method is not adapted to all cases, and does not always effect a radical cure.

2. *Tying the artery*. This may be done in various ways: (1) close above the aneurism (Anel); (2) higher up above the aneurism at a locus electionis (J. Hunter); (3) close below the aneurism (Wardrop and Brasdor). Of all these methods, that of tying the artery immediately above the aneurism is comparatively the most certain, immediately below it the most uncertain. In placing the ligature at a distance above the aneurism, a cure will be effected for some time, sometimes also permanently, *i.e.* the pulsation in the aneurism will cease, but if the collateral circulation become sufficiently established, the pulsation in the aneurism may recur. I have seen one such case myself; a boy about twelve years old had had an aneurism of the femoral artery, about the size of a large walnut, form at the middle of the thigh in consequence of a puncture with a penknife. The artery was tied immediately below Poupart's ligament; ten days afterwards the ligature came away with a considerable amount of hæmorrhage, which was soon checked, however. A second ligature was now applied half an inch higher up, after

dividing Poupart's ligament. This ligature remained firm, the wound healed, but when the patient left the hospital, fresh pulsation could be detected in the aneurism, which had become quite hard after being tied, and had ceased to pulsate. In spite of such relapses, tying the artery at a distance from the aneurism will still retain its value and continue to be the chief method, because the arteries in the immediate neighbourhood of aneurisms are sometimes so much diseased that it is not advisable to tie them there. The rigid and calcified arteries may, namely, be so rapidly cut through by the ligature that the thrombus is not firm enough, when the ligature comes away, to offer resistance to the pressure of the blood.

3. *Remedies of which it is assumed that they cause directly coagulation of the blood in the aneurism.*—Of these the injection of liq. ferri sesquichlor., according to Pravaz and Petrequin, has come comparatively most into use recently; it must be employed very cautiously. A small syringe is used for this purpose, fitted with a screw which forces out one drop at a time when turned. This small apparatus is fitted to a very fine, sharp-pointed canula, which can easily be pushed into the aneurism. A few drops of the liq. ferri are introduced very cautiously therewith into the tumour. Simple coagulation and shrinking of the aneurism may and are said to be caused by this, but experience shows that inflammation, suppuration, and gangrene more frequently follow this operation. I believe that a mistaken view is held of the effect of the injected liq. ferri; there is, namely, very little probability that a coagulum imbued with liq. ferri becomes organised, but the walls of the vessel are probably irritated and inflamed, and thereby become incapable of keeping the blood passing along them in a fluid state, so that coagulation and shrinking of the vessels are caused secondarily only. V. Langenbeck injected into the immediate neighbourhood of aneurisms a solution of ergotine, and effected cures therewith; I explain to myself the effect of this treatment also as consisting in inflammation of the walls of the vessel followed by the consequences just mentioned.

Electro-puncture, little thought of for some time, has now again been employed, with very favorable results, by Ciniselli, even for aneurisms of the aorta. The method consists in introducing a needle into the aneurism, and bringing the negative pole of a galvanic battery into communication with it, while the positive pole is placed in contact with the body at some point. It was thought formerly

that the galvanic current possessed the property of causing the blood to coagulate directly; physiological investigations have shown that this is not the case, but a small eschar is formed by the thermic action of the current around the needle introduced into the aneurism, and upon this the coagulum forms. If several fine needles are thrust into the aneurism, and left twenty-four to twenty-eight hours *in situ*, inflammation of the vessel and coagulation are also caused; this mode of proceeding is called *acupuncture*.

4. We now come to that treatment of aneurisms which has for its object the *complete destruction* thereof; if this succeeds, it is of course more certain as regards a radical cure than all the methods of treatment previously described; but, as an operation, it is much more exhausting. We may perform it as follows on Antyllus's plan; the trunk of the artery is to be compressed above the aneurism; the entire sac is now to be laid open, the coagula removed, a probe introduced from the cavity into the upper and lower end of the artery, and both of these ends tied. The probes are then removed, as they were merely intended to assist the operator in finding the artery easily and quickly. This operation, which I have performed several times, is not always so easy as it might appear, because it is sometimes rather difficult to find the opening of the artery in a sac filled with coagula; other arteries than the main branch frequently bleed, because collateral branches also sometimes open into the aneurism. After the operation, suppuration of the whole aneurismal sac sets in. In many cases of traumatic aneurisms of the femoral, brachial, and radial arteries, I have seen a cure effected without any interruption. If the aneurism is small and very distinctly defined, it might first be tied above and below, and extirpated as a tumour. The method of Antyllus has been successfully tried in spontaneous aneurisms of very large arteries by Syme. There can be no doubt that it will be more frequently tried now that we can, thanks to Esmarch's method, empty of its blood the limb on which we wish to operate, and do the operation more quickly and more surely than heretofore.

With such various methods of operating to select from I should like just to give you a few definite rules for your future guidance, and yet this is scarcely possible, because, according to the different individuality of the cases, first one method and then another has to be preferred. I can only repeat generally that as such good results have again recently been reported from different quarters of compression, this

method ought not to be too early given up. But if, as is usual in traumatic aneurisms, there is a diffuse swelling, it appears to me that the method of Antyllus is the most preferable of all—it is certainly the safest, and would be the best were it not for the danger of hæmorrhage at the seat of the ligature. Ligature, according either to Anel or Hunter's methods, must be done when we cannot or will not operate according to Antyllus' plan. We should more quickly determine on ligature not only of large arterial trunks, but also of the smaller vessels, were it not, as I have already said, for the fact that extensive hæmorrhage so frequently occurs at the seat of the ligature. Perhaps some day we shall invent a method of occluding arteries which will possess all the advantages of the ligature without any of the disadvantages. Of injection of perchloride of iron, either in spontaneous or traumatic aneurism, I would say least. In varicose aneurism and in aneurismal varix ligature of the artery above and below the opening is the surest method.

It is necessary to add a few remarks on the treatment of cirsoid aneurism. The above-mentioned operations are only partially applicable to this disease. Direct compression of the whole tumour can be made by bandages or by some specially contrived compressorium; I refer now more especially to cirsoid aneurisms on the scalp, where they oftenest occur. Compression, however, has scarcely ever succeeded. The injection of perchloride of iron might be tried in these cases, because sloughing or gangrenation of the whole arterial loop is not so very formidable, as in aneurisms of the large trunks of the extremities. A short time ago I cured a cirsoid aneurism on the forehead by means of acupuncture. Obliteration might also be accomplished by destruction of all the afferent vessels; this is, however, very troublesome and of uncertain value. Equally uncertain and not without danger is ligature of one or both external carotids in cirsoid aneurism on the head. This treatment ought to be discontinued. Another method, which accomplishes the same purpose, is to pass needles in different places beneath the tumour, and then to apply silk ligature thread in figure of 8; sloughing and obliteration take place, and possibly also partial gangrenescence of the skin. Total extirpation is the surest method; it may be accomplished thus:—we make a number of subcutaneous punctures all round the tumour, close to each other; then the chief portion, with the dilated arteries, can be cut away without hæmorrhage. This is also the surest and most radical operation, but is not applicable in

tumours of great size, though we might remove portions of the tumour at different times, and thus finally eradicate it. Heine, as the result of his own very thorough researches on the treatment of these aneurysms, speaks strongly in favour of complete extirpation.

LECTURE XLIII.

CHAPTER XXI.

TUMOURS.

Limitation of the term Tumour. General anatomical remarks. Polymorphism of tissue-forms. Sources of origin of tumours. Restriction of cell-developments to particular kinds of tissue. Relation to development. Mode of growth. Anatomical metamorphoses in tumours. External appearance of tumours.

GENTLEMEN,—To-day we commence the difficult chapter which treats of tumours. When we have hitherto spoken of swellings, they were only such as might be caused either by abnormal collections of blood in and around vessels, or by infiltration of the tissue with serum, or by its permeation with young cells (plastic infiltration), or by some combination of the separate causes just given. In contradistinction to these *swellings*, the new growths clinically are called *tumours*; for the most part they result from other and unknown causes than those which give rise to inflammatory new growths; they possess a power of growth which, as a rule, comes to no typical termination, but which, within certain limits, may go on indefinitely; and besides this, tumours consist of tissue, which is very completely and more permanently organised than the inflammatory new growths. Let us consider this a little more carefully. At present you are only acquainted with that variety of neoplasm which is caused by inflammation; we found that this inflammatory new growth was not only very uniform in the nature of its formation, but also in its further development; it might be limited in its development by disintegration, drying up, or suppuration; it might proliferate inordinately, but it would be in such a way as not to change its character; lastly, however, if there were no unfavorable local or general conditions, and if the organ affected was not essential to

life, the new growth might become absorbed, or it might become connective tissue—in other words, *the inflammatory process terminated in the formation of cicatrix.* To this, in superficial inflammations, was added the development of epithelium and epidermic cells through the epidermis, the bony cicatrix ossified, and in the nerve cicatrix new nerve-fibres were formed, and the formation of new vessels in all the cases played an important rôle. But, as we before said, the process of inflammation, whether acute or chronic, superficial or deep, has its most typical termination in the formation of cicatrix. Now, although connective-tissue, nerve, and bone tumours may occasionally form out of connective-tissue, nerve, or bone cicatrices, they nevertheless form but a very small proportion of the varieties of tissue found in tumours. Forms, the most varied and complex—*e. g.* newly-formed glands, teeth, hairs, &c.—are sometimes found in tumours; tissues may even be found which occur nowhere else in the body nor in fœtal life during the period of development.

In order that you may have a correct idea of the anatomical nature of tumours, I will just recall to your minds a few general ideas as to the formation of new growths. You will find excellent and complete descriptions of these conditions in the exhaustive works on this subject by Virchow and O. Weber. When a part of the body is abnormally enlarged, we first of all decide whether the enlargement is due simply to an abnormal increase *in volume* of the individual elements (simple hypertrophy), or to the formation of *new elements* deposited among the old ones. This new growth may be analogous to the diseased mother-tissue or matrix (homœoplastic) or the contrary (heteroplastic). The homœoplastic new growth proceeds either from simple subdivision of the existing elements (for instance, one cartilage-cell by division becomes two, and by further subdivision four, and so on); this is *hyperplastic new growth* (numerical hypertrophy); or from the existing cell elements are formed small, round, apparently indifferent, cells, which then develop into tissue analogous with the mother-tissue; this is *homœoplastic new growth* in the stricter sense of the word. *Heteroplastic new growths* always begin by the development of primary cell formations, so-called indifferent formative cells (the granulation stage of tumours according to Virchow), from which is developed a tissue heterologous to the matrix, for instance, cartilage in the testicle or muscular fibres in the breast.

This classification, proposed by Virchow, from a purely anatomical point of view appeared both natural and convenient, and I can still accept it, provided the idea of heteroplasia be limited in the manner to be shortly described, and provided also the idea of identifying homœoplasia with benignancy and heteroplasia with malignancy be completely given up. We must also consider whether the wandering cells, escaping from the vessels, assist in the formation of tumours, at all events of those tumours outside the connective-tissue series. But, apart from these considerations, we must not suppose that all classes of new growths, even considered solely from an anatomical point of view, could be thus easily classified, as though on the numbered shelves of a museum. Simple numerical hypertrophy and hyperplasia, though in some cases, perhaps, difficult to distinguish, can, theoretically at least, be easily separated; the same may be said of those new growths which entirely consist of similar, well-formed elements. A tumour consisting of connective tissue occurring in connective tissue is always homœoplastic; occurring in bone, the brain, or the liver, it would be described as heteroplastic, and so on. Well-developed alveolar cancer presents no difficulties for its classification, because it does not occur anywhere in the body under normal conditions, and it is, therefore, heteroplastic. But what are we to say of new growths which do not show any well-developed normal and yet no very abnormal type, but which seem to consist of elements of which it is impossible to say, whether or how they develop further (indifferent formative cells, wandering cells, primitive cell-tissue, granulation-tumours). Further, where shall we class those new growths which, though they do not consist of fully developed (perfected) tissue, are found to resemble certain well-known types of the fœtal stage of normal tissue? According to this view of heterology and homology, inflammatory new growth is everywhere heterologous at first. Very well; but later on the connective-tissue cicatrix becomes homologous, if it develop in connective tissue, while it remains heterologous if it occur in muscle, or in the brain, or even in bone unless it ossify. Thus you see that that which, according to its nature and mode of origin, is naturally allied, becomes separated by this anatomical classification. But let us leave inflammatory new growth out of the question! Every tumour which proceeds from indifferent cells must, if the cells are converted into tissue or into a variety of tissues, go through a series of developmental stages. The indif-

ferent formative cells are everywhere heterologous when heaped together; if a new growth show only these elements, let us call it heterologous; but if it also show that a quantity of these cells has been converted into spindle-cells, it becomes necessary to enquire now where does this neoplasia belong? We may say spindle-cells, when collected together in quantity, are everywhere heteroplastic; but yet spindle-cells occur in fœtal connective tissue, in fœtal muscle, and in fœtal nerves. What would have become of these spindle-cells eventually? Is not the tumour, even if we find it in muscle, to be called homologous? On this point we can only decide arbitrarily. It may be looked at from different points of view. What, then, shall be said of tumours which contain the most diverse tissues, both of perfect and of imperfect formation? I will, however, leave off, in order not to make you too sceptical; I am here to facilitate your studies rather than to make them more difficult.

Since the enlargement of the individual elements (simple hypertrophy) cannot be observed, and the increase of the elements out of themselves (hyperplasia) is an act often observed and constantly going on in physiological development, it only remains for us to consider the *source of origin of the indifferent formative cells*, and their further destination. Here again we find ourselves in the same position as in inflammation, though unfortunately, as regards the development of tumours, we cannot undertake any experimental investigations. Formerly no one doubted that every kind of connective-tissue cell could proliferate, and this was looked upon as the source of development of most tumours; at present we are bound to admit that many of these indifferent cells may be wandering blood-cells. In this matter, therefore, we have been in error by hastily endeavouring to draw conclusions as to their origin from the mode of their arrangement in groups and their metamorphoses. Nor can I myself claim exemption from these errors. If, for instance, small indifferent cells, with one, two, or more nuclei, were found close together—if between the fibres of connective tissue, where the connective-tissue cells lie, a group, at first small, then larger, of indifferent cells was seen—the conclusion seemed obvious that the groups of new cells were the offspring of the connective-tissue cells, and that from these indifferent cells others, constantly increasing in size up to the giant cells (so-called), should be formed. Now, however, we are aware that this small-cell infiltration of tissue may

result from the migration of the white blood-cells from the vessels into the tissue, and hence we feel more doubtful as to the origin of the indifferent formative cells in tumours. Of late I have sought, and especially in glandular and epithelial cancers, for proliferating connective-tissue cells, and generally in vain, although the entire connective-tissue stroma of these tumours is generally infiltrated with young cells. The great obscurity which until a very short time ago surrounded the origin of the youngest epithelial cell has only very lately been cleared away. As the result of the most recent investigations, we now know that these cells increase by a kind of *budding out* (Sprossenbildung). I must here again refer to my remarks on the regeneration of tissue during the process of inflammation. We must remember that, according to Arnold's observations already alluded to, possibly in the formation of tumours, protoplasm, completely converted into tissue, again degenerates into a granular condition, and forms within itself nuclei, and then begins to proliferate; a 'budding-out' of tissue, analogous to the 'budding-out' of a cell, takes place; then, when the granular protoplasm has differentiated itself into cells, the formation of new tissue results. Hence the doctrine of Schwann, according to which all tissue results from cells is not prejudiced, though the doctrine "omnis cellula ex cellulâ" is thereby considerably modified. We have constantly spoken of indifferent formative cells, without having precisely defined the term: by them we understand the small round cells, which are everywhere found in tissue that has been irritated, and with which we first became acquainted in inflammatory new growths. Until within a few years I believed that these young cells really were as *indifferent* as the earliest segmentation cells of the ovum, that is, that any and every tissue of the body might finally develop from them. I thought more especially that from the offspring of the connective-tissue cells, not only all forms of connective substance (connective tissue, cartilage, bone, vessels and nerves), but also epithelial structures and glands, might be formed. This view, first enunciated by Virchow, is still held as correct by many investigators in this field. Thiersch, however, in his excellent work on "epithelial cancer," has opposed such weighty proofs against this view that I feel obliged to entirely agree with him. As I intend to return to this subject when speaking of cysts, glandular tumours, and epithelial cancer, I shall here only give the merest outline of my present views. The doctrine of development teaches

us that the body of the young embryo very early shows three separate, so-called, embryonal layers. As soon as this differentiation of the cellular elements into the three germinal layers is accomplished all observers agree that then each of these layers can produce only a certain series of tissues. Thus, from the horny layer are formed the nervous system, the epidermis and its derivatives, the cutaneous glands, the sexual glands, the labyrinth of the ear, and the lens; from the middle layer are formed the connective tissues, the muscles (?), the vascular system, the lymph-glands, the spleen; and from the undermost or glandular layer the endothelium of the intestinal tract and of the lungs, and the secreting elements of the liver, pancreas, kidneys, &c. This seems to be some natural law, for the discovery of which we are particularly indebted to Remak, Reichert, Kölliker, His, Waldeyer, and others, which may possibly be referred back to the composition of the egg itself. In the whole subsequent course of development it never once happens that a tissue develops from the derivatives of one germ layer which were originally formed from those of another. In other words, when the differentiation of the rudiments of an embyro into its three layers has once taken place there no longer exist indifferent cells, but all new cells originating from the former old ones can develop only into those tissues which come within the area of production of that layer in which they originate; cells which come from true epithelium can never produce connective tissue, and those from the derivatives of connective tissue can never become true gland-producing epithelium. There is no reason to suppose that this natural law ceases to prevail when the cellular elements of a finished organism are stimulated to production by any irritation; the young brood can only develop into certain pre-ordained tissue types, which depend upon the embryonal source of the mother-cells. *Heredity is the most powerful law of living nature.* Thus, whenever we have spoken or speak of indifferent cells we must always limit the signification of the expression by the application of the above principles. If we now return to the earlier plan of new growths, as taught by Virchow, according to our view there is no real heteroplasia, for the cells resulting from the derivatives of one germinal layer can only further develop within certain limits of tissue type, and can never be transformed into the tissue types peculiar to another germinal layer. Owing to the vigorous movement which is going on on the subject of histiogenesis, any too

absolute assertion is in danger of being obliged to submit to some or other modification; but I must, nevertheless, repeat that it appears to me to be exceedingly probable that a very large proportion of the young cells, which escape so extensively into the tissues during the development of tumours, are movable, wandering, connective-tissue cells, that is, escaped white blood-cells. I do not, however, deny to the fixed connective-tissue cells all participation in the formation of new tissue. Something has already been said of the participation of muscular and nerve tissues, of cartilage cells, epidermis, and epithelial cells in the formation of tissue. It is still uncertain how the wandering cells get into the blood (they are identical with white blood and lymph cells); they probably form from the fixed elements of the lymph glands and spleen; at all events, they are to be considered as elements of the middle germinal layer, and their eventual power of development must be regarded as limited to tissues which spring from this layer. We may look with pride at the progress which morphology has made in recent times, the importance of which is shown by the fact that it has been so destructive to previous views, and yet so fruitful in the most varied directions.

It has been especially remarked by some investigators in this subject that the above-described conditions of fœtal development cannot be considered as irrefutable natural laws, and that they only apply, as a rule, in the development of the more highly organised animals. I must leave this point, however, to be decided by embryologists. I would, nevertheless, strongly reject the doctrine that modes of development which are known to obtain in embryology do not merit acceptance for the new formations which develop in adult tissue as the result of irritation; for the whole doctrine of modern histiogenesis is based on Johannes Müller's suggestive work on tumours, in which he laid down the now generally accepted principle that the development of pathological new growths is only a repetition of the typical development of normal tissues. We should loose our hold, and relapse into the old chaos of parasites (παρά, near, and σῖτος, food) and pseudoplasms (ψεῦδος, false, πλάσσω, to form), if we were to let go of this principle.

Let us now return to tumours proper. The life and growth which unfold within them may be very manifold. In the first place the diseased portion of tissue, the first tumour-nodule, may grow in itself, without the occurrence of fresh disease, in the vicinity

of this focus: in the centre of the tumour, and from the cells heaped up at some circumscribed spot, fresh cells develop, which possess the same developmental tendencies, and which are, as it were, predestined for the particular type of tissue of which the new growth consists. It was formerly believed that vascular dilatation was a very important symptom of inflammatory new growth. Numerous researches in this direction have convinced me that the dilatation and new formation of vessels during the development of the first tumour-nodules is not less, either in extent or importance, than in inflammation. At the same time it has not yet been shown that any softening alteration of the capillary and venous walls, similar to that which occurs in inflammation, ever takes place. The original focus of disease may also spread by the continuous formation of fresh foci in its immediate neighbourhood; an organ once diseased in this manner is not only pressed upon by the tumour and its elements separated, but the organ itself becomes more and more diseased by the appearance of fresh foci (of disease), and thus becomes infiltrated and destroyed by the tumour, and finally absorbed in it; for you have already learnt that where new growth occurs in the normal tissues, the matrix, as such, ceases to exist, and in part becomes converted into the new tissue, and is in part destroyed by it. Thus, in the first case we have an isolated disease-focus, which, once there, provides for its further development entirely from its own cells; while in the second case it obtains them from new secondary foci, which constantly spring up in the immediate neighbourhood of the first. This first kind—this, so to speak, *central growth*—is very much less dangerous to the diseased organ than the latter, the *peripheral growth*; for should the latter continue to progress, it must necessarily, by infiltrating the tissue with new growth, lead to the entire destruction of the affected organ, just as does an inflammatory process, or inflammatory new growth when it continues progressive. The most unfavorable condition is the combination of these two kinds of growth, which, unfortunately, is not at all rare. If we further study the life-history of a tumour, we find that the newly formed tissue does not by any means remain stationary; it is subject to many changes, some of which are also observed in inflammation. Acute or chronic inflammations may occur in tumours from various causes; that is, a small cell, even purulent, infiltration with wandering cells in the tumour-tissue may occur, accompanied

with pain, swelling, and vascular dilatation, the result of inflammatory disturbance in the nutrition. Tumours, in which the cell formation is so excessive, and progresses so rapidly, that the formation of vessels cannot keep pace with the growth of the tumour, are least capable of living; slight disturbances then suffice to interfere with the process of development, or, as we cannot altogether arrest it, to bring about its destruction. We must go a little more thoroughly into the metamorphosis of tumour-tissues. They may be either acute or chronic; acute inflammations of tumours are, on the whole, rare, though injuries, blows, or contusions may give rise to them.

This traumatic inflammation in vascular connective-tissue tumours may terminate in resolution with or without cicatricial shrinking; more frequently it is followed by more or less extensive extravasation, gangrene, or suppuration. Chronic inflammations in tumours are far more frequent; not only such as are characterised by the exuberant production of inflammatory new growth and the formation of fungous ulceration with extensive vascularisation, but also those which are characterised by slow ulceration. Caseation and fatty degeneration of the tissue, also mucoid degeneration, are not very rare. In these softening processes thrombosis of the vessels and collateral plugging around the softening mass take place, just as in those retrogressive inflammatory changes which lead to abscess or caseation. In consequence of the processes just alluded to, connected with the development and disease of tumours, the anatomical characters may become so obscured that it may be impossible at first sight to recognise the kind of tissue of which the tumour was primarily composed. Finally, it sometimes happens in the course of time that the anatomical characters of tumours change; for instance, a connective-tissue tumour which has retained its character for a long time may suddenly become very soft in consequence of a rapid cell proliferation and vascularisation, while, on the other hand, a soft tumour may become hard owing to absorption of its cellular elements and shrinking of the connective tissue within it. Thus, you see that a considerable amount of knowledge and experience are necessary, in order to rightly appreciate in individual cases even the anatomical characters which form the basis of the doctrine of tumours; it will sometimes happen that it is quite impossible to give to the case we have examined a name by which it may be classed in any of our given groups. As to the nomenclature

of tumours composed of different kinds of tissue, we must name it after that kind of tissue which is present in the largest quantity.

There is now a general inclination to append the affix "ὤμα" to the corresponding tissue in order to give it a histological definition:—thus sarkoma, carcinoma, &c. The word "ὤμα," however, did not exist in Greek; it has been obtained in this manner: the affix "οω" was added to certain nouns in order to make them into verbs; thus σάρξ, flesh, σαρκόω, to make flesh, καρκίνος, cancer, καρκινόω, to make like cancer, to bend or crumple. The Greeks made use of the expression σἄρκωμα, flesh-tumour; καρκίνωμα, cancer-tumour (Hippocrates). The modern nomenclature has been built up on this type, and it is to Virchow especially that we are indebted for it. The old Greek term for tumour in general is "ὄγκος," which signifies crooked, bent, rough. Hence the term also used by Virchow, "onkology," the doctrine of tumours. Another expression, sometimes used by Hippocrates, is φύμα, φυτόν, a growth, but now seldom used. The term "struma" (from struere, to lay one upon another, to build up), formerly used by Celsus to signify swelling in general, and then especially swellings in the neck. The English still retain the expression. That which we (the Germans) call "lymphatic" or "scrofulous" they call "strumous." At the present time the word "struma" is used (in Germany) to signify exclusively tumours of the thyroid.

I have but very few remarks to make on the external naked-eye characters of tumours. In most cases they are roundish nodulated growths, more or less easily distinguishable both to the sight and to the touch, from the surrounding structures. This is not always definite, however; tubercles also, certainly in their earliest stages, are rounded, circumscribed, though bloodless structures, which I should, however, no more class among the tumours than papules or pustules of the skin. A growth may occur in the skin as a definitely formed nodule just in the same way that an abscess may form, which also at first appears as a nodule. Still, as chronic inflammatory new growths on the surface not infrequently appear in the form of papillary proliferations (villi), so also a growth which forms in the skin may assume a papillary form: the surface of a tumour also, or the interior of a sac or cyst, containing fluid, may independently give rise to papillary proliferations. Thus you see that the purely anatomical conditions of tumours or of inflammatory new growths do not suffice to accurately define their exact limit.

A variety of terms exists to express the different qualities of tumours, which are even now frequently used, although they do not always apply. Thus, a tumour situated in a cavity and attached by a pedicle is called a polypus (from πολύς, many, and πούς, a foot—centipede), and hence we have nasal, uterine polypi, &c.; then we have to add its histological peculiarities, as fibrous, sarcomatous, myxomatous, &c. Growths which have ulcerated, and project like a fungus, and resemble one in form also, are called spongy or fungous, yet the same term—spongy or fungous—is applied to the sponge-like softening of diseased tissues. When we desire to say that a tumour is very rich in vessels and blood, we append the word "hæmatodes," or "telangiectatic" (from τέλος, end; ἀγγειõν, vessel; ἔκτασις, dilatation), or "cavernous." If a tumour was very hard or fibrous (not bony or cartilaginous) it used to be called "scirrhus" (σκιῤῥός, hard). This term is now no longer used in this sense, and still less its corresponding adjective, "scirrhous," which, after all, only signified hard, and was applied to inflammatory induration just as much as to cancer. Medullary is applied to a tumour which has the colour and consistency of the brain, while its structure may be that of either a sarcoma, or carcinoma, or lymphoma; and as tumours of this appearance are known to be especially malignant, they go by the name of "medullary sarcoma," "medullary carcinoma"—terms which are also freely used to designate any kind of malignant growths without any reference to their structure. Many tumours are pigmented—light brown, yellow, brown-black, or blue-black; these pigments may have resulted from hæmorrhages, or be due to some specific cell activity. "Melanomata" (μέλας, black), or melanoses, are rare, consisting either entirely or partially of black or brown-black pigments, and in structure belong either to the sarcomata or carcinomata. Their prognosis is usually very unfavorable. Formerly surgeons were content with these and similar terms, and with comparisons to one or other of the normal tissues; suffice it for you to be acquainted with the meaning of these expressions.

Now that you somewhat better understand the subject, we must once again come back to the term "tumour." Pure anatomy should simply reject this term; for it there are only new formations (organised neoplasia, Rokitansky), either simple or compound; from a series of observations it can show how these growths originate, and what becomes of them. But we do not thus arrive at the meaning

of the word "tumour" in the sense in which it is used in pathology. A tumour, or growth, in the modern pathological sense, conveys a definite ætiological and, for the most part, prognostic signification; it is, as we showed at the commencement of this section, a new growth, which, as a rule, is not due to any of those causes which give rise to inflammation, but rather to others, for the most part unknown or but dimly suspected. The process, which produces in the organism, either locally or generally, tumours, is generally considered quite distinct from the inflammatory; the two processes are believed by many—with what right we do not venture to say—to stand in direct antagonism to each other. Though we cannot deny that in any given case causes which generally produce inflammations (traumatic, thermic, or chemical) may not sometimes stand in an etiological relation to tumours, yet we should consider this as a so very unusual case that we should incline to regard the constitution as itself peculiar. This, what I venture to call the pathological and physiological, view has not been previously maintained, but I do not think I am mistaken in stating that the doctrine is now held, consciously or unconsciously, by most pathologists. All writers on tumours avoid the subject as much as possible, because there is nothing more to say on it. We do not know where or how to draw the line between the etiology of chronic inflammations and the production of tumours. It is just as little possible to give the term "tumour" a purely anatomical definition as it is to explain the disease "typhoid" anatomically; we are compelled to fall back on a compromise between ætiology and pathological anatomy. In the etiological signification of the expression "tumour-forming process" is included the idea of the ultimate fate of this product—a tumour—which is quite other than that of the inflammatory new growth. Hence we may say of tumours that they do not contain within themselves the conditions necessary to typical resolution as do inflammatory new growths. For my own part I cannot think that the inflammatory process stands in any really antagonistic relation to the tumour dyscrasia; rather would I think that clinical observation teaches us that the two processes in many cases unite together, especially in some forms of chronic inflammation and sarcoma growth, while, on the other hand, an acute metritis and an uterine fibroid lie far enough apart both etiologically and anatomically. It is not seriously disputed that the formation of tumours is due to specific causes. Virchow believes that it results from a powerfully inflammatory

diathesis; thus polypi of the mucous membranes result from long-continued catarrh; syphilis first produces inflammation, and then tumours. I will here just mention that I do not class syphilitic products among tumours; a gumma, or caseous nodule, resulting from syphilis, heals up by absorption or after incision and suppuration—a circumstance which only occurs excessively seldom in a tumour. H. Meckel von Hemsbach has advanced the contrary opinion; he says, for instance, that enchondroma of the finger is the mildest outcome of a scrofulous diathesis, and corresponds to pædarthrocace on the fingers, &c. If we compare the products of inflammation with the more histologically developed forms of tumour, we must confess that tumours, as being more slowly developed, are due to a more feeble irritation than inflammation, and are more allied to normal growth. The considerations before mentioned all apply to true tumours only, of which we shall alone speak. Virchow, by including as tumours encapsuled blood extravasations and dropsies of serous cavities, places himself quite beyond the bounds of our present views.

LECTURE XLIV.

Ætiology of tumours. Miasmatic influences. Specific infection. Specific reaction of irritated tissues; the cause is always constitutional. Internal irritation. Hypotheses as to the nature and mode of action of irritation. Course and prognosis. Single, multiple, and infectious tumours. Dyscrasia. Treatment. Principles of classification of tumours.

Let us now more minutely consider the ætiology of tumours. It is thus that we may hope to find out the differences as well as the relations of those processes which give rise on the one hand to inflammatory new growths, and on the other to tumours. Let us commence with the causes of inflammation, and then compare them with those of tumour formation. Many acute inflammatory processes (exanthemata, typhus, &c.) and some chronic ones (intermittent fever, scurvy) result from miasmata and contagia, which enter the body from without. I cannot admit of acute miasmatic tumours; but the development of goitre may be considered as a miasmatic, chronic tumour. We cannot well regard goitre as the result of an inflammatory process, since goitre never resolves spontaneously nor suppurates, nor cicatrises. Their cause is, however, a specific one, coming from without, and to which every individual, and especially young individuals, are exposed who come into a district in which goitre is endemic, although all are not equally disposed to it, for there is probably some hereditary tendency. The infection is probably conveyed by the blood; it is difficult to imagine that the thyroid gland can be influenced locally by miasmata. Hence, then, goitre is probably the local expression of a general infection, which sometimes shows itself in the general nutrition of the body, and sometimes in an anomalous development of the skeleton resulting, cretinism. The Oriental disease, elephantiasis, may be considered as a chronic miasmatic condition, in which large masses of knotty fibrous tissue form in the skin on different parts of the body, generally associated

with anæsthesia. I am bound to admit, however, that in this instance it is somewhat disputed, and that reasons may be advanced which would seem to indicate that the disease does not belong to the tumour diathesis, but must rather be classed among chronic inflammations. As regards local infection, or the conveyance of fixed contagia from without, we already know that inflammatory processes of various kinds are produced. Inflammatory processes are alone produced by means of decomposing fluids; here I include also the so-called post-mortem tubercle, which I cannot consider as a tumour, because it will disappear spontaneously as soon as no fresh inoculations take place. By inoculation with the pus of certain stages of an inflammation, another inflammation is set up, and according to the nature of the pus will be the specificity of the process; pus may also give rise to a general disease, which would then manifest itself by the presence of multiple localised inflammations, as in syphilis for instance. Can we give rise to tumours by inoculating with the juice of tumours, or with small portions of tumours? This is still a disputed point. I, personally, consider it possible, though not absolutely certain. The difficulty of determining lies in the fact that it is not possible to perform such experiments on man; and if similar experiments from man to animals fail, it only proves that human tumours are not inoculable on animals. The tumours of animals must be engrafted on to animals, and at present the only experiments which have been made are those by Doutrelepont, who inoculated the cancer of dogs on to dogs, but without any result. However, it is quite certain that inoculation with pus will not produce tumours, which again seems to point out the specific difference of these products. Some pathologists will probably reply that molluscum contagiosum is an instance in which the juice of tumours or portions of tumours is successfully inoculated on to other persons. This fact, established by Ebert and Virchow, is exceedingly interesting; nevertheless, the contagiousness of this new growth is for the present too isolated to allow us to draw any general or reliable conclusions from it. The clearest proof of the specificity of inflammatory products and of tumours is afforded us by the observation of the local and general infection which we have daily the opportunity of making in cases of inflammation and tumour formation.

We have already often spoken of progressive and secondary inflammations, of the almost always secondary (deuteropathic, Vir-

chow) acute lymphangitis, of the secondary acute and chronic swelling of the lymph glands in acute and chronic inflammations especially of the extremities. While on this subject I said to you that I considered it probable that cell elements from an inflammatory centre found their way into the lymph glands, and produced in these glands, by virtue of their specific phlogogeous properties, inflammations which are analogous to primary peripheral inflammatory processes. Such-like local inflammatory infections never result from the presence of tumours, and when the original inflammation has subsided, the swelling of the lymph glands also subsides; in chronic glandular swelling the subsidence does not always take place so rapidly. Similar infectious qualities also belong to many tumours, and especially those which, like inflammatory neoplasia, are multiple; for not only may the immediate vicinity be infected and numberless small foci formed around the primary tumour, but also the lymphatic glands may become affected, and secondary deposits may occur in them, which will possess the same attributes as the parent tumour. They are, too, just as unlikely to disappear or subside as the latter, even although the primary tumour be removed; on the contrary, other secondary tumours of a similar nature may occur in distant parts of the body, hence called metastatic tumours. Here we again have the analogy with the course of inflammatory infection, and yet the specific differences; for metastatic tumours never result from phlogistic infection, and just as little do metastatic abscesses of nternal rgans result from tum our infection. All tumours are not infectious, although unfortunately the vast majority are. Infectious tumours are called malignant in contradistinction to the non-infectious, which are hence called benign. It is very difficult to say in what this difference consists; it no doubt lies partly in the specific characters of the cell-elements, in their easy motility, and in the fact that, like the semen of many of the lower plants, these elements everywhere find a suitable soil in which to germinate in the various tissues of the body, and thus grow into tumours themselves; it also partly lies in the fact that the conditions for the taking up of the tumour elements by the lymph channels or by the blood-vessels is sometimes more, sometimes less favorable: thus, for instance, it is remarkable that very soft tumours, consisting almost entirely of cells (medullary sarcomata), if surrounded by a fibrous capsule, do not affect the lymphatic glands; the same may be said of many encapsuled abscesses. I have already remarked to you, concerning metastatic

abscesses, that they are all of embolic origin; but as regards the diffuse metastatic inflammations we must look for some other explanation. Diffuse metastatic tumours, however, are excessively rare; indeed, I could only recognise a few pleuritic or peritoneal carcinomata or sarcomata as such.

As to the mode of formation of metastatic tumours—that is, the real course of the infection—we may judge by analogy that, like secondary tumours of lymph glands, they spread by means of semen from the primary or from the secondary tumour. I acknowledge that I am much inclined to accept this view. Although at one time I could not bring myself to believe that the cells from an inflammatory focus or from a tumour could be as independent as the spores of the algæ, yet I must now admit that, with our present knowledge of the independent life of pathologically formed cells, we can no longer doubt the possibility of such a process. Quite recently, indeed, some observations have been made known which afford the highest evidence of a high degree of independence, which the tissue elements, especially the cells of the rete Malpighii, possess — I mean the oft-mentioned transplantation of epidermis of Reverdin. According to this there is still stronger reason to believe that detached cellular elements of a new growth, carried away either by the lymph or blood-stream to different regions of the body, may there take root and grow.

If on the first formation of a tumour, or of an inflammatory new growth, the lymph vessels should become partially closed and filled with cells, it is quite possible that in the further course of the disease lymph and vascular thrombi should be caused by this stenosis, into which the specific tumour elements may wander, and then minute particles of thrombus, such as are formed in the process of softening, may be carried into the circulation first here and then there, and so give rise to further growths. The formation of such thrombi filled with specific tumour elements has actually been observed in larger and smaller veins, and analogous tumours have likewise been found in the branches of the pulmonary arteries. This circumstance is worthy of remark, that metastatic tumours, like metastatic abscesses, are especially found in the liver and lung, except in these cases, where the transference of the tumour metastasis is direct, as in pleural tumours, which occur secondary to tumours in the breast, or in liver tumours, which occur secondary to primary tumours of the intestine or stomach. In these cases a direct transference

of the tissue elements along the lymphatics is very easily conceived of. On this subject there is still room for investigation, and I believe it would lead to successful results. The products of acute inflammation mostly act, as we have already seen, as pyrogens; those of chronic inflammation possess these qualities in about the same small degree as tumours, for in the latter only when disintegration takes place, and its products get into the circulation, does fever occur. If we review what has been said concerning the contagiousness of tumours, it results that there is a possibility of transferring the specific elements of a tumour from one individual to another, though it is not yet proved absolutely; but it is now proved beyond doubt that by means of the lymphatic glands various kinds of tumours may be propagated, and that other organs in the same individual may become implicated. There are various views on the manner in which this secondary affection takes place. The simplest is that already referred to, viz. that the detached emboli of a vessel blocked up by the primary tumour themselves take on growth, and that the neighbouring tissue comports itself as when in the presence of any other foreign body, with this exception that it sends-in vessels into the embolus. Others believe that the tumour affects the neighbouring tissue in such a manner that it produces tissue similar to that of the tumour. Others, again, rather fancy that the juice of the tumour alone suffices to excite an ordinary tissue to the production of tumour tissue. In speaking of the development of carcinoma we shall have to refer to this subject again.

Concerning local and general catarrh as a potential cause of inflammation, there seem to be no observations which would warrant us in referring the development of tumours to analogous causes. I am not aware that any one has ever proved or even asserted that tumours might result from cold.

Very various views are held concerning mechanical and chemical influences, as causes of tumour formation. Although very various irritations exist, and although many experiments have been tried, there is at present not one single instance in which a tumour has been produced as the direct result of mechanical or chemical irritation; the inflammatory new growths which have resulted in such cases have never long outlived the cessation of the irritation. Whenever and wherever we apply mechanical or chemical irritation, we always produce simple inflammation. So that if specific

mechanical or chemical irritants (I mean such as act on the organism from without, and not emanating from an already existing tumour) really do exist, that is, such as *must* necessarily give rise to a tumour, then they are not as yet known. The next question is, Are there grounds for assuming that such specific mechanical and chemical irritants outside the organism necessarily exist? I cannot concede this; truly there are cases in which a tumour has developed after a blow, or contusion, or a wound, but the number of cases is strikingly small in proportion to those in which after a similar injury, either a typical, acute, traumatic inflammation, or after a longer continued irritation, a chronic inflammation with typical course has resulted. We must, therefore, regard this as the rule. Thus, if a porter gets a thickening, and beneath that a newly formed bursa over the spines of his vertebræ, or a sore in the same position, we regard it in some measure as a natural process—they are the result of a chronic inflammatory irritation, and they subside immediately the irritation ceases. But if an individual get a fatty tumour in the same place as the result of similar causes, which, however, does *not* disappear but even continues to grow after all irritation has been removed, we do not regard the irritation as a specific one, but rather seek for the peculiarity in the tissue itself. We have already acknowledged the specificity of irritants in general and local infections; now we must also acknowledge that the tissues possess a specific and qualitatively abnormal reactionary power. Virchow and O. Weber have especially shown that local irritation from without plays an important part, especially in the development of primary tumours; and this is well shown in the fact that primary tumours most frequently occur in parts of the body at which external irritations are most common. Thus, statistics show that the stomach is the most frequent seat of tumours, then the portio vaginalis uteri, then the face and lips, then the mammæ, then the intestine. That tumours, however, and not inflammations occur in such cases, must be due to the *specific predisposition* of the part in certain individuals. People who take much spirits get gastric catarrh; if in a thousand drinkers, one or even ten, instead of catarrh, got cancer, they would be considered as abnormal subjects compared with the remainder. Thus far I agree entirely with Virchow, who expresses himself on this point as follows: "Although I cannot explain in what special manner an irritation must act in order, in a given case, to produce a tumour, and in another, and under

apparently similar circumstances, to produce a simple inflammation, I have communicated a series of facts which teach that certain continuous disturbances may exist in the anatomical structure of some parts, which interfere with the occurrence of certain regulating processes, and that these parts, when subjected to an irritation which in other places would only produce a simple inflammation, then become the seat of specific tumour-formation. Among the facts "which teach that in an anatomical structure of some parts certain continuous disturbances may exist" which predispose to tumour formation, Virchow mentions advanced age: it is perfectly true that certain forms of tumour in given localities are especially frequent in advancing life, as, for instance, cancer of the lip. Thiersch draws attention to the fact that in the lips of old men the connective tissue has extensively disappeared, and in consequence the epithelial structures (sebaceous, sweat, mucous glands, and hair follicles) become more prominent, and receive an overshare of the nutrition; hence there occurs a preponderance in the growth of these epithelial structures, a fact which will well explain the frequent occurrence of epithelial cancer in the lips of elderly men. I willingly admit the shrewdness of these observations; but I feel bound to remark, nevertheless, that advanced age is just as much a general as a local attribute of the body, and therefore it cannot be regarded as a local predisposition. Virchow further states that "places which have previously been the seat of an inflammatory disease (in consequence of which the part becomes weakened), and also scars serve as foci for the development of tumours. This is undoubtedly true; but yet if one compare the numberless instances in which chronic inflammatory changes simply occur, although these places may have been the seat of acute mischief, we shall find that the number of cases where tumours occur in these places is remarkably small, and that in these few individuals a specific predisposition, leading to the formation of tumours, may be assumed. The same may be said of organs which only acquire their full growth and development in later life, and which are known to be peculiarly prone to the development of tumours. Virchow also here mentions the articular extremities of bone (which, however, are the seat of tumours much more rarely than of chronic inflammations), the mammary glands, the uterus, the ovaries, the testicles. While fully appreciating the thought and observation which have been displayed in attempting to prove

a purely local predisposition to the development of tumours, I cannot myself accept these proofs as at all convincing, and for the present, at least, continue to believe that *there is a specific predisposition to the development of tumours just as definite as that which leads to chronic inflammations either with the proliferation of the inflammatory new growth, or with suppuration, or with caseation, &c.*

We must add to what has already been said that we cannot at all constantly prove the existence of a local external irritation in the case of tumour development, any more than we can in the case of local disease in scrofulous subjects. While I must refer you to what has already been said on the ætiology of chronic inflammations, I would just remark, in relation to the development of primary tumours, that in many cases we do find within the body itself, certain specific so-called internal irritations. Most pathologists will grant this, though the source and mode of action of such irritants is variously explained. Virchow teaches strongly that local disease must have a local cause, and assumes that certain conditions of local debility exist at the point of disease. According to this we should have to allow a specific local debility for the most varied disturbances of nutrition as well as for tumour formations. Rindfleisch expresses himself concerning internal irritation very decidedly as follows: "As the result of tissue change there are constantly being formed certain excretory products which must be got rid of, not only from the tissues and organs in which they are produced, but also from the fluids of the entire body, if the processes of life are to go on without interruption. The substances take their chemical position midway between the organic nutritive bodies on the one side and the excretory products of the kidneys, skin, and of the lungs on the other; they fall into the great gaps which exist in organic chemistry at this point. They vary slightly in each case for the different tissues, and on this difference rests the peculiarity of pathological new growths. If they are not properly transformed and excreted, they accumulate first at the spot where they are formed, then in the juices of the organism, and this accumulation is the immediate cause of the setting up of the progressive processes which begin by a simple multiplication of cells in the connective tissue, and end by the formation of either tubercle, cancer, epithelioma, fibroma, or lipoma, &c." I can thoroughly subscribe to this view, but I must add that it appears to me that we deceive

ourselves in speaking too especially of local processes. The secretion of bile and of urine certainly are local processes, but for them to be produced in a given quantity or of a given quality depends on so many conditions which are outside the glands, and which appertain to the entire organism, that one may truly say, the primitive cause of the secretion of either bile or urine must be sought not in the blood only, but in the entire organism, or even in idiosyncracies as remote as Adam, if you so please.

In a like sense I believe that the primary causes of the local conditions which lead to the development of tumours must be sought in the specific attributes of the individual organism. Thus we speak of a scrofulous or of a tubercular individual, and by this we mean the pathological race to which, as I have said, that individual belongs.

I must finally just add that the belief that the irritation, the cause of the disease, is exactly localised at the spot where later on the tumour forms, is just as hypothetical as any that have yet been advanced. Let us take arthritis as an analogy. Zaleski has produced the most typical arthritis in a goose by ligaturing the ureters; joint disease in consequence of a disturbance of the renal function. It is just as possible that tumours might develop in some one of the tissue systems as the result of liver disturbance! Here anything is possible. We know nothing for certain on this subject, all being hypothetical. For my own part I think it is just as allowable to assume a diathesis for tumours as for scrofula, or arthritis, &c.; and that partly from unknown and partly from known causes in connection with general nutrition and the ordinary conditions of life, certain abnormal products result, which act specifically on this or on that part, according to the analogy of certain drugs. Let us add to this that the tumour-producing diathesis is hereditary, although in a lesser degree than the diathesis which produces chronic inflammations; the doctrine of localised weakness of one or other system or of one or another part of the body appears to me untenable. That the members of one branch of a family have large noses certainly has a local cause; they may have grown disproportionately to the rest of the face as compared with other persons, but the large nose of the father cannot be inherited as such; it can only come through the spermatozoa of the father, and there the original cause must be looked for; all qualities that are inherited must undoubtedly be called constitutional.

I have long occupied you with reflections which to some must certainly appear tedious, and such will ask me what use will they be in practice? I must unfortunately admit that practice takes very little heed of such things, just because they are so very hypothetical, and occupies itself with much more concrete observations. Let this comfort you, that those of you to whom such ideas never occur need never trouble about them. For, not to be *obliged* to speculate on the ultimate cause of things is, in a certain sense, an enviable lot.

Let us, for the sake of easier reference, briefly summarise in a few sentences what we have said on etiology.

Tumours, like inflammatory new growths, result in consequence of irritation of the tissues: the difference of the predisposing cause lies (1) in the specific nature of the irritants. Of this fact the infection of healthy neighbouring tissue and of the nearest lymphatic glands is accepted as sufficient proof. It is accepted hypothetically also that, under some unknown circumstances, specific irritant matters may be formed in the tissues at the diseased spot (Rindfleisch). I am of opinion that, partly in consequence of inherited and partly acquired predisposition, that is to say, as the result of a diathesis, the development of materials in the organic juices is conceivable, which act specifically and irritatively on one or other tissue. (2) Any, and for the most part, inflammation-producing irritants can give rise to a tumour, provided always that the tissues so irritated possess the specific predisposition to tumour formation. Virchow, O. Weber, Rindfleisch, and others believe that these specific qualities are quite local and confined either to a limited area of the body which may be accidentally irritated, or else to a system such as the bones, the skin, muscles or nerves. But for my own part, I cannot conceive such a localisation of specific attributes as possible, and therefore I conclude that in this hypothesis the apparently local specific qualities are to be explained by idiosyncracies which stand in the closest relationship with the rest of the organism.

You see from this *résumé* that the difference in the views held lies solely in the hypothetical part. If I have entered into the subject more exhaustively than appeared necessary for these lectures, my excuse must be that this subject, so important to general pathology, has been very thoroughly and clearly treated of by Virchow, O. Weber, Rindfleisch, Lücke, Thiersch, Waldeyer, and

others, and I have thought it the more necessary to develop my own views on those points where I differ from the above-named authorities, whose excellent writings I cannot too strongly recommend to your most earnest study.

As regards the prognosis and course of tumours, you will gather from what has been said (1) that tumours do not tend to spontaneous cure, and that they are not amenable to drugs; and (2) that some are infecting, and some non-infecting. This last point is especially striking for the uneducated mind. There are tumours which do not recur after extirpation, and others which not only recur in the scar of the operation and in its immediate neighbourhood, but also, in the course of time, in the nearest lymphatic glands, then in internal organs, as has already been said. The first have for ages been called benign, the latter malignant tumours, or cancer. This observation appears so simple that it would seem to be only necessary to study the nature of one or other of these varieties, in order to be able to give a definite prognosis in any given case. An accurate clinical and pathological study unfortunately, however, does not lead to such a much-to-be-wished-for result, but it rather teaches that such a sharp demarcation does not exist at all, and that the facts are much more complicated. After having thoroughly exhausted all the external anatomical points, one examines tumours with the microscope and in the retort, and is led to believe that first one point and then another is the chief characteristic; after a while another discovery proves the other one false, and finally we learn that there is no absolute division between malignity and benignity in the above sense; we learn that there is something to distinguish besides solitary, or multiple, or infectious tumours; that there are, indeed, *degrees* of infecting power also. We must consider this more closely. We speak of a tumour as *solitary* when only a single one occurs on the body at one time; they consist for the most part of a highly formed tissue—fibromata, chondromata, osteomata, and so on. We speak of *multiple* tumours, if a series of similarly organised growths occur on one tissue system, as, for instance, chondromata only in connection with the bones, or many lipomata only in the subcutaneous tissues, or many fibromata in the skin. This is caused, and it is now generally acknowledged, by a predisposition of the diseased system, which Virchow regards as purely local, but which I think must be

referred to a general constitutionary dyscrasia. Generally speaking, we may say that tumours may occur either single or multiple, although the latter is an exceedingly rare occurrence for some varieties. We call tumours *infectious* when they not only infect, but also grow into the neighbouring parts, infiltrate them, and by the apposition of new centres of growth spread; they affect the nearest lymphatic glands too, and, after a time, disturb parts and organs of the body. In this respect there are very great differences; thus, in some forms of cancer the infection travels regularly to the nearest cluster of lymphatic glands (cancer of face or lip), in others it goes further, especially to internal organs (cancer of the breast), while in another set of cases infection of the whole body takes place, and metastatic tumours form, without any infection of the lymph glands, as in many sarcomata. Besides this, the rapidity with which the infection spreads is very remarkable. If we examine the conditions under which infectious tumours develop, and the anatomical structure of such growths, we shall find that they occur especially in advancing age, in men and women about equally, and particularly in certain organs. Early childhood is very prone to infectious growths, especially malignant sarcomata; while youth and early manhood seem tolerably exempt from tumours, and especially so from infectious ones. The mode of life, good or bad nourishment, poverty, opulence, character, nationality, the influences of education seem to exercise little or no influence on the development of tumours. Neither can we conceive of any specific influence being exercised by these potentialities on the development of infectious tumours.

The study of the microscopical structure of tumours has latterly been pursued with great zeal, and the result is that a large number of the infectious tumours has been found to possess macroscopic as well as microscopic characteristics; but they do not allow any certain prognosis to be based on them. As a general thing, it may be said that they are very rich in cells, disposed to ulcerate, and in due course prove infectious. As it is exceedingly probable that infection takes place from the locomotion of specific cell elements, the factors in absorption must be duly taken into consideration. The amount of blood- and lymph-vessels in the tumour and its immediate neighbourhood, the conditions which regulate the opening and shutting of these channels, the force of the circulation, especially, are all factors which must be duly considered.

Infectious tumours are generally solitary at the commencement, scarcely ever multiple in the sense just described. Tumours which from the very commencement are multiple are seldom infectious. When we use the terms dangerous, malignant, and infectious synonymously, we do so without regard to the position at which the tumour develops. A single benign tumour, if it occur in the brain, is nevertheless *quoad vitam* always malignant, on account of its position; an infectious tumour in the same position never gets beyond a local infection, because it kills too soon. All these things must be carefully considered if we would get clear views on the subject.

Tumours are not always to be called infectious (malignant, cancerous), simply because they recur locally after an operation. For in such cases, it must be decided whether the recurrence has taken place from parts of the original tumour which were left behind at the time of the operation (continuous recurrence of Thiersch), or whether, after complete removal, a fresh growth has occurred either in the scar or in its immediate neighbourhood, which is due to causes similar to those which produce the original growth (regional recurrence). If the seat of the primary operation remain quite free, but if growths, similar to the original tumour, take place in the lymphatic glands (infective recurrence), or in internal organs, it is safe to assume that the lymphatic channels were infected at the time of the operation, even after an examination failed to detect the fact.

If an individual is infected by or from a tumour we call it dyscrasia, just as we call a person who is affected from an inflammatory centre dyscrasic (pyæmic). In such individuals foreign matters are circulating in the vital juices, which induce in them pathological characters; this dyscrasia manifests itself, in the case of infectious tumours, by a general disturbance of the nutrition, emaciation, and marasmus; as to the extent to which it proceeds depends chiefly on the seat of the growth and their characters (softening, gangrenescence, ulceration, hæmorrhage, &c.), as also on the amount of strength and on the age of the affected person.

Concerning *the treatment* of tumours I shall here only mention that they are alone curable by removal from the body, either by the knife, the ligature, the écraseur, or caustic, or other means. The removal of virulent and rapidly infectious tumours is, at most, but a means of prolonging life and of mitigating the sufferings of

the patient. In the case of non-interference, the treatment must be symptomatic, and aim at lessening the suffering of the patient. I will give the indications for operation when speaking of the individual forms of tumour.

In passing on to the consideration of the different *varieties* of tumours, we almost shrink from the mass of material which lies before us. We need a leading principle to guide us in the arrangement of tumours so various, both pathologically and clinically, and in their mutual relations to each other and to the organism at large. The principles according to which tumours have been classified are as various as the principles on which general diseases have been and even now are classified. Not one system of classification of disease which has been hitherto invented has survived very long. Pathology is now taught in many groups of smaller systems, and the principles on which these groups are selected are manifold. Before pathological anatomy had reached its present development it was usual to seize on certain prominent symptoms, and thus, in medicine, we have still such terms as jaundice and apoplexy, &c., and in the doctrine of tumours there are expressions, well known to you all, as polypus, scirrhus, fungus, carcinoma, &c. As soon as the symptoms, jaundice and apoplexy, resulting from many different pathological causes, were analysed these terms were rejected from the nomenclature, and the expression of the actual pathological condition substituted.

The anatomical and pathological nomenclature of diseases as arranged by Rokitansky is undoubtedly highly scientific; so, too, the system of general pathology by Virchow; and yet neither one nor the other has been absolutely adopted by clinicians. Some wished to classify diseases according to their peculiar nature and causes; but Schönlein's attempt to found a system on this plan failed because our knowledge of the causes and of the nature of diseased processes did not suffice to fully carry out this idea. What has been the result? Practical medicine and surgery rest on an anatomical basis, assume this as generally known, and make use of it for the subdivision of those larger groups of diseases, which are founded on ætiological, prognostic, symptomatic, or physiological bases. It would not be unscientific now-a-days to write a monograph on jaundice or apoplexy. In such a case the anatomical conditions would come in the second rank, and pathological anatomy,

like other scientific aids such as chemistry and physics, would also be made use of; we constantly keep in mind that our object is to understand the diseased process as a whole rather than its morphological details ; we strive, in fact, to grasp not only the pathological processes, but also the nature and cause of the physiological disturbance. It would, indeed, be unscientific in the case of typhoid fever, even if we do find a number of palpable changes, to wish to see nothing but a peculiar kind of intestinal inflammation, and we may now venture to regard this as something of the past. If we could group all diseases from an ætiological point of view it would be an immense advance; pathological physiology could then take the place of pathological morphology. With our present knowledge we are proud to be able accurately to recognise the morphological developments of the diseased product, because then we can say that we are acquainted with at least one of the important factors of the pathological process. In truth, however, we are not one whit advanced in the knowledge of normal development, and it will be long before we understand the physiology of the growing fœtus.

After these considerations we cannot pretend to a classification of tumours any more than of diseases generally. We must acknowledge that there will be differences according as whether we select ætiology, symptomatology, prognosis, or morphology as our guiding principle. Surgeons in the past have preferred to classify tumours, according to the prognosis of the individual case, into malignant and benign, and, in addition, have made a few sub-sections according to the outward form of the growths, or their consistence or their appearance on section. This sufficed so long as the observation of these points was made wholesale, and the surgeons did not make any great pretence at prognosis. However, when observation at the bedside became more accurate and tumours assumed more various forms under the microscope, it became more and more impossible to reconcile the anatomical characters of tumours with the older views on malignity and benignity. While now most surgeons and pathological anatomists relinquished the idea of allowing prognosis to take any part in the classification of tumours, and since Johannes Müller's works on this subject directed chief attention to the constant working out of the minuter anatomical and developmental details of pseudoplasms, I myself endeavoured still to retain those so-clinically prominent symptoms of benignity and malignity in a wider sense as the principle of classification of tumours, and to

render them subordinate to the most recent acquisitions of pathological histology. Whether I did not adopt the proper form and expressions for my ideas, or whether the task which I had set myself is really beyond accomplishment, I know not; but at all events I am alone in my views on this subject, and hence I have given them up; and even if to-day I am still of the opinion that we ought not to cease in our efforts towards a physiological (ætiologico-prognostico-clinical) knowledge of the processes which determine the formation and growth of tumours, and though I should even now value a classification of tumours on a physiological and genetic basis still more highly than one on an anatomical and genetic basis (from which Virchow, in his wonderful and classical work on tumours, set out), nevertheless I cease from further attempts in this direction, and follow the anatomical principles of division, by proceeding from tumours composed of simple tissues to those which consist of more complex structures.

I must finally mention that I shall confine my lectures arbitrarily and designedly to those cases of tumours which, at their commencement at least, are situated on parts of the body that are within reach of the surgeon. This limitation is not of such vast importance as it at first seems to be; it can be well imagined that we can only study the peculiar course of tumours when they are localised in parts which are not directly necessary to life, for the symptoms which accompany or supervene on liver or stomach or brain tumours, for instance, are not due to the presence of a tumour as such, but are the result chiefly of a disturbance in the function of the affected part. If every case of typhoid were accompanied with fatal hæmorrhage or perforation of the intestine, we should never get a true type of the ordinary disease because of the interference with its natural course. We shall here and there give indications as to the relative frequency of primary localisation of tumours in internal organs, but we cannot enter into the symptomatology and histology of the diseased organs; on this point you will be instructed by your pathologists and teachers of clinical medicine.

LECTURE XLV.

1. *Fibromata.*—A. *soft*, B. *firm. Mode of onset; operations; ligature; Ecrasement; Galvano-caustic.* 2. *Lipomata; anatomy; onset; course.* 3. *Chondromata; onset; operation.* 4. *Osteomata; forms; operation.*

1. *Fibroma, fibrous tumour—Connective-tissue tumour.*

TUMOURS, which for the most part, consist of well-developed connective tissue, we call fibromata. The following are the varieties:

A. *The soft fibrous tissue, or connective-tissue tumour.*—They occur rather frequently, and are generally found in the cutis; they consist of a very tough, rather œdematous, white tissue, and are generally covered by a thin papillary layer of the cutis. A microscopic examination shows loose connective tissue, as in the cutis, and on the surface of the tumour almost always pointed papillæ, even when the tumours occur in places where the cutis normally is not covered with papillæ. In the rete Malpighii of these growths a brownish pigment is frequently found, seldom deeper in the tissue; they may also contain large vessels, and abnormally hypertrophied hair follicles and sweat-glands, on their surface. They are usually loosely hanging and often well pedunculated growths (cutis pendula, molluscum fibrosum). They may also be regarded as partial (local) skin hyperplasias, as they chiefly consist of the elements of the cutis. Their growth is very slow, absolutely painless, and it may proceed to an enormous size. Sometimes these tumours are congenital; they may be multiple; hundreds of such tumours may occur on the surface of the body. Congenital cutis proliferation occurs mostly on the face; it is generally one-sided, and either diffuse or localised in the form of cockscomb-like vegetations. The larger moles—hairy nævi with pigmentation (mouse-skin, benign melanoses, melanomata, pig-

mented fibromata)—belong to this class. These tumours develop most frequently at the end of middle life; in women it is not uncommon to find pendulous tumours of this kind on the labia majora, and as tumours on these parts are concealed as long as possible, they are generally very large before they come to the knowledge of surgeons. Virchow terms the disease, which tends to the production of these multiple, soft, fibrous tumours, Leontiasis. In the course of time they occasionally give rise to general disturbance of the nutrition. Although these growths are not infectious in the sense already referred to, they nevertheless do sometimes lead to a cachectic condition, and in the course of years to death from marasmus. There is an anatomical relationship between this disease and so-called "Elephantiasis Arabum," although by this term we generally refer to a more nodulated, though at the same time diffuse hypertrophy of the cutis (cutis pudenda) of certain parts of the body (scrotum, leg), which occurs after recurrent erysipelas. It would certainly lead to misunderstanding if we were to call this simple hypertrophy of the skin, or pachydermis. Elephantiasis Græcorum is, as concerns the thickening of the skin, a similar though strongly endemic general disease, accompanied by nervous symptoms (hyperæsthesia and anæsthesia and imbecility; it occurs in Greece, Asia Minor, and Norway (under the name of Spedalsked), and after a protracted course generally leads to death.

B. *Hard fibromata, fibroids, and desmoid tumours*, to the naked eye seem to consist of a firm, closely-woven fibrous tissue. They are always of a very firm consistence, of rounded, knotty form, and in section either pure white or pale red. Some of them on their surface present a very peculiar, regular, and concentric arrangement in layers around distinct axes (Fig. 125). According to my obser-

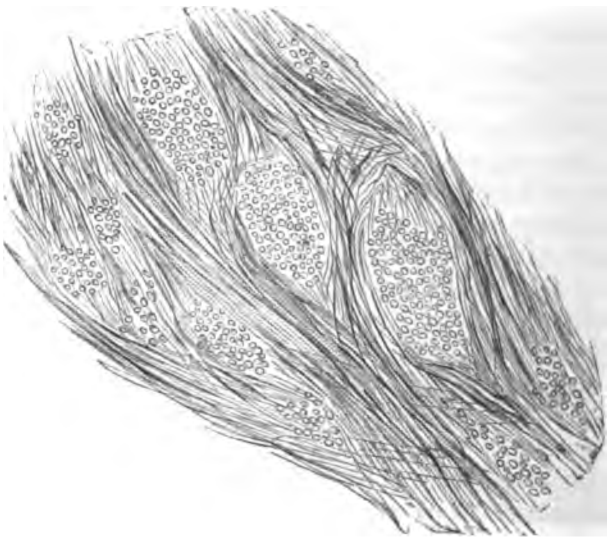
FIG. 125.

Small fibroma (myo-fibroma) of the uterus; natural size of the cut surface.

vations, this is due to the fibrous formation taking place around nerves and vessels, the latter being consequently embedded in the centre of the fibrous layers; not infrequently the nerves are by this means destroyed.

With the external characters just described the histological appearance renders it difficult to assign a place to these tumours in the list. There can be no doubt that such of them as consist chiefly of firm connective tissue, as, for instance, all old uterine fibroids, must be called fibromata; younger tumours, however, with a similar appearance and consistence, shows very little connective tissue, but abundance of spindle-shaped cells.

FIG. 126.



From a myo-fibroma of the uterus. Magnified 350. Vertical and longitudinal sections of the muscular bundles.

The interpretation of these cells varies. Virchow considers them to be muscular fibres, and hence he classifies the tumours which have hitherto always been called uterine fibroids and classed among the fibromata, among the myomata, and calls them myoma lævicellulare. If we consider these fibre-cells as young connective tissue, we shall have to christen them spindle-celled sarcomata or fibro-sarcomata. Thus, you see, even in the case of simple fibrous tissue we are already in difficulties with histology and histogenesis. There are two reasons which would induce me to regard these fibro-

cellular tumours as myomata, viz. the remarkably club-shaped, undulating form of the nuclei and the definite arrangement of the fibrous layers into bundles, and the difficulty of isolating the individual fibres except by the aid of the recognised chemical means. In addition to this, the locality in which the tumour is developed must be carefully borne in mind; the probability of its being a myoma becomes very great if the new growth is found in the substance of the uterus.

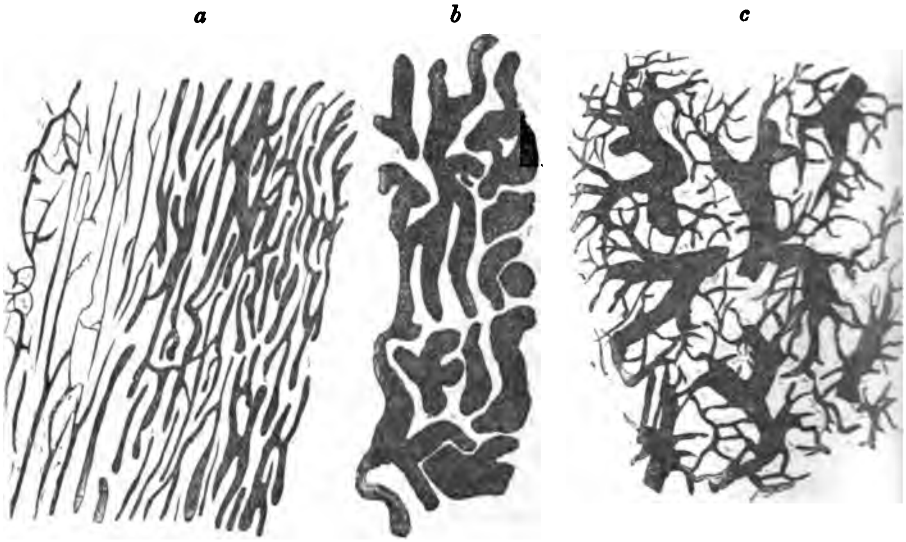
Fibromata are capable of certain pathological metamorphoses; partial mucoid degeneration, great serous infiltration, brawny appearance and consistence, calcification, and even true ossification, may not infrequently be seen in them. Superficial ulceration is somewhat more frequent in those fibroids which lie close beneath a mucous surface; it results from external injuries in the usual manner. Such a sore may present healthy granulations and suppuration, and under favorable conditions it may quite heal up. Fibrous tissue, though apparently poor in blood-vessels, nevertheless does contain some, as may be proved by injecting them; in some cases they contain many vessels, both arteries and veins. Occasionally an extensive network of veins may form within them (Fig. 127). Arteries and veins are so intimately united with the tumour tissue that this adventitia becomes blended, so that in case of injury they cannot retract either lengthwise or transversely, and their lumina then remain gaping. This is the mechanical and pathological explanation why hæmorrhage from fibrous tumours is so profuse, and why it cannot, even with skilled help, always be brought to a still-stand. The rigid, gaping lumen hinders the formation of thrombus in the highest degree.

We sometimes find in the large uterine fibroids, and also in those connected with periosteum, lacunar spaces filled with thin serum; perhaps these are pathologically dilated newly-formed lymph sinuses. Definite observations on this point do not exist; cavities as large as a bead and filled with serum also occur in uterine fibromata (Spencer Wells).

The position of fibromata varies greatly; of all the organs, the uterus—provided in the term fibroma we include also the myofibroma—is most frequently affected; here these tumours sometimes reach an enormous size, and then sometimes calcify. Generally they are rounded in shape, they are clearly and sharply separated from the surrounding parts, they generally occur in the body of the

organ, more rarely in the cervix, scarcely ever on the lips of the portio vaginalis; they usually grow upwards or downwards, that is, either towards the abdominal cavity, with gradual stretching of the peritoneum, or through the orificium vaginale into the vagina. In

FIG. 127.



a and *b*. Vessels of a cutis fibroma (myoma?) from the thigh, injected from the artery. *b*. Cavernous veins. *c*. Regular and curiously branched veins of a cutis fibroma (myo-fibroma?) from the abdominal wall, injected from a vein. Magnified 60 diams.

the latter direction, these tumours grow more and more, are pedunculated, and often give rise to serious hæmorrhages. They are called fibrous polypi of the uterus.

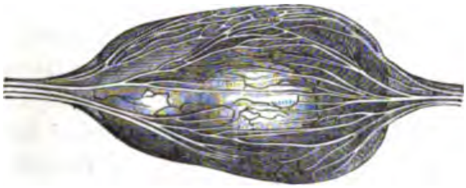
Fibromata growing from the periosteum are very common; they are almost always fibro-sarcomata, that is, they are made up of fibres and spindle cells, and the latter sometimes predominate (fibrillated sarcoma of Rokitansky). The periosteum of the skull and of the bones of the face are very liable to this disease, and especially the under surface of the turbinated bone. Fibromata here appear as polypi in the nasal cavities, and in the pharynx (fibrous naso-pharyngeal polypi).

They may cause absorption of the bone by pressure, and may even grow into the skull or into the antrum of Highmore; they are particularly rich in cavernous veins. Besides these, I have seen

fibromata on the periosteum of the tibia, and also on the clavicle, and even in the interior of bones, in the upper jaw, for instance, where I have also met with remarkable combinations of chondroma and fibroma.

Finally, we must mention that both in and on nerves, small as well as great ones, fibromata not infrequently occur (Fig. 128).

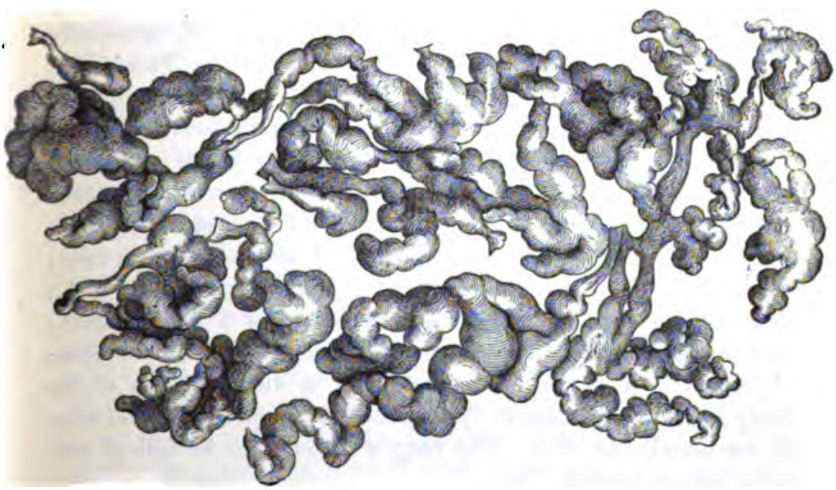
FIG. 128.



Neuro-fibroma, after Follin.

All tumours occurring in connection with nerves are mostly called neuromata, but they ought to be distinguished according to their anatomical characters. Most neuromata (so-called) are fibromata, or fibro-sarcomata on the nerve trunks, while a few contain, or even consist entirely of, newly formed true nerve fibres, and are then true neuromata. Sometimes nerve-fibromata follow the nerve trunks, and form knotty cords (plexiform neuroma, Verneuil, Fig. 129), on a confluence of which, as already stated, the peculiar appearance of the cut surface of some fibromata occasionally depends (Fig. 125). They are generally congenital.

FIG. 129.



Plexiform neuro-fibroma of the cheek, after P. Bruns. Natural size.

The development of fibromata in the subcutaneous cellular tissue belongs to the rarities; in the glands, except in the breast, they scarcely ever occur. The fibrous tumours just described are peculiarly liable to occur about middle age (from thirty to fifty years); they seldom occur in youth, and almost never in advanced life; if we occasionally find them in the uterus of an aged woman they have probably been there for many years. Fibrous neuromata and bone and periosteum fibromata only occur in young persons. In general, fibromata occur somewhat more frequently in women than in men. Uterine fibroids develop between the thirty-fifth and forty-fifth years, although it may be much later before they give rise to much trouble; they are more often multiple than single. Periosteal fibromata are generally single; they may recur even after the course of many years (regional recurrence—relationship with sarcomata). Fibromata generally grow from the centre; they are not infectious, though infectious fibromata *are* said to occur; a number of such tumours near each other may coalesce, and infiltrate the surrounding parts, and occasionally cause fibrous degeneration of the nearest muscles, bones, and lymph glands. The infectious fibromata which I have seen were all fibro-sarcomata, and like pure sarcomata, they may occur metastatically in the lungs. Neuro-fibromata are very frequently multiple, especially in different branches of the same nerve. A short time since I extirpated six of these tumours from a man; three from his left arm, and three from his left leg. Cases are on record of twenty to thirty neuromata existing together. Pure fibromata generally grow very slowly, and in old age they sometimes cease to grow. This is best known of fibromata of the uterus, which may cease to grow after the period of involution and then calcify. Combinations with other forms of tumours, mostly with sarcomata, as before remarked, occur, and generally in such a way that the primary tumours show a fibrous character, while the recurrence and the secondary tumours are found to be soft, cellular sarcomata. I have seen such a case; a man aged about twenty-five years, of healthy appearance, had a fibro-sarcoma on the abdominal wall as big as a large walnut; the tumour was completely extirpated, but in the wound there appeared another tumour, and later on in different parts of the body several soft tumours; the patient became marasmic, and after a few months he died. The lung was found to be full of soft sarcomatous tumours.

After what has been said, the diagnosis of fibroma is not difficult; consistence, locality, mode of attachment, shape of tumour, almost always lead to a correct and sure diagnosis.

Treatment, to be effective, must consist in removing the growths. If at all, this must be generally done with a knife; but pedunculated or pendulous tumours, and fibrous polypi, allow of other modes of operation. In such cases the ligature was formerly much used; the pedicle of the tumour was tied tightly with a cord, so that the tumour became gangrenous, and sloughed, and finally dropped off. This method was especially chosen in those cases where hæmorrhage was to be feared. Ligature has this disadvantage, that the tumour in or on the body gangrenesces, and that it has to be retied in a number of cases before it finally cuts through. In such cases, hæmorrhage of a severe nature may occur. The ligature and the knife may be combined; so that after the ligatures have been tightly adjusted, the bulk of the tumour can be cut off, and only a portion of the pedicle left to slough. In the nasal and pharyngeal cavities, as also in the vagina, there are naturally great difficulties in applying a ligature; a great number of instruments, both simple and complicated, have been invented—so-called snares, by means of which the thread is passed over the tumour on to the pedicle. At present the ligature is generally rejected, and so little used that all these instruments—some very clever, indeed—are now for the most part only of historical value.

The desire to remove pedunculated growths without hæmorrhage is still very strong, and it has in recent times led to the introduction of new methods, and of instruments, the use and adoption of which have only been possible since the use of chloroform has become general. Crushing and the actual cautery have now taken the place of the ligature. We have already referred to Chassaignac's *Ecrasement*. By means of this operation, if carefully performed, there is no hæmorrhage, even from arteries the size of the radial. The wound thus made is smooth and sharp, and heals perfectly without much sloughing on its surface. And although in all cases hæmorrhage cannot certainly be avoided, it is so in most. The instrument has been constructed in all sizes; the smallest can be used very conveniently for the nose, and small pedunculated polypi can be easily removed with it. Another method, but with similar action, is the galvano-caustic, introduced into surgery by Middeldorpf. It consists in heating a platinum wire noose (connected

with the two poles of a galvanic battery) previously placed around the pedicle of a tumour, and burning through it. The result is a separation and an arrest of hæmorrhage at the same time. As for the arrest of hæmorrhage, it fails about as often as in écrasement—on the whole very rarely; the method, therefore, is for certain cases much to be recommended. The trouble to the surgeon of preparing a strong, useful battery (which is very expensive) is such that galvano-caustic does not at the present moment seem to have much prospect of being at all largely adopted; in spite of its elegance it has been eclipsed by the introduction of the écraseur; the medical public has already expressed its verdict; most operating surgeons possess an écraseur, while galvano-caustic apparatus are only found in a few hospitals.

As regards the operation on the non-pedunculated, more deeply seated fibromata, in many instances the tumours are quite beyond surgical help. Although in some cases Köberle, Péan, and others have removed uterine fibroids through the abdomen with the greatest success, I would, nevertheless, not yet venture to recommend this practice, not only because the operation is very dangerous, but because such tumours, in the course of time, sometimes cease to grow, and because the troubles they give rise to seldom outweigh the danger to life of the operation. Concerning these fibromata, which are not dangerous to life, either from their position or mode of growth, the operation on which, however, would be dangerous, we must always remember that their growth is slow, and in later life often ceases altogether; hence one must not hastily undertake such cases or urge them too forcibly. There still remains a large number of other cases for which an operation may and must be undertaken; especial indications for operation are persistent and dangerous hæmorrhage from an ulcerated fibroma, threatened disturbance of bone, pressure into the skull, and the like. In cases of neuro-fibroma the pain is sometimes so severe that the patients themselves urgently ask to be operated upon, even after they have been told that a paralysis of the corresponding muscles will be the inevitable result, for in such cases it is almost always necessary to remove a portion of the affected nerve, which may still either completely or partially perform its functions. If neuromata be painless, it would be a mad undertaking to excise them.

2. *Lipomata. Fatty Tumours.*

A disposition to the formation of fat, if it do not exceed a certain degree, is, of course, not considered as a morbid diathesis, but rather the reverse, and as a sign of unusually good nutrition. It varies considerably with the age, and is generally most developed between the thirtieth and fiftieth years; it occurs more rarely in childhood; it is favoured by a quiet, regular life and a phlegmatic temperament. We only regard this as a disease when a disturbance of the function of some organ or of the whole body is brought about, and when the formation of fat is confined to a single region of the body, when in fact it forms a fatty tumour.

The anatomical characters of a fatty tumour are simple enough: it consists of a fatty tissue, which, like the subcutaneous fat, is divided into lobules by strands of connective tissue. These connective-tissue strands may be more or less developed, and hence the tumour may appear more or less firm (fibro-lipoma), or soft (simple lipoma). Their external form is generally round and lobed, and they are separated from the neighbouring structure by a thick sheath of connective tissue (hence circumscribed lipoma, the ordinary form); and they are easily separated from their surroundings. More rarely lipoma comes on as a growth confined to one especial part or member of the body, and then appears as a swelling without any definite boundary (diffuse lipoma). I once observed a case in which the newly-formed fatty tissue had involved and grown in between the muscles of the right thigh of a young girl, so that the operation, which I had commenced, could not be completed.

The seat of lipomata is most frequently in the subcutaneous cellular tissue, especially of the trunk, and mostly on the back or abdominal walls: lipomata on the extremities are rarer; an enormous production of fat may occur in the synovial folds of joints and in the sheaths of tendons, so that the fat masses may assume a branched appearance something like a tree (Lipoma arborescens, J. Müller). This is in analogy with the proliferation of fat in the processes of the peritoneum of the large intestine (appendices epiploicæ), and other serous membranes. This variety is extraordinarily rare. The growth of lipomata is very slow, and their development is quite unaccompanied with pain, unless they are

closely connected with nerve-trunks, and either stretch or press upon them, which very rarely happens. Fatty tumours may attain an enormous size; the patients, little troubled by their presence, seldom feel inclined to have them removed while small, and so they are allowed to grow until they become enormous tumours. A short time since I removed a lipoma from the back of a woman, which was attached to the right scapula, and hung down as low as the calf of the leg. At its base its circumference was about that of the upper part of a thigh, while its lower extremity measured twice as much. Secondary changes are not usual in these tumours, though they may certainly occur: the thicker connective-tissue strands in the tumour calcify, or even ossify, while the fat tissue may turn into an oily or emulsion-like fluid. The skin covering these tumours is gradually expanded, and at first generally thickened; at the same time it is rather pigmented brownish, and the papillæ increase in size. The skin generally continues movable over the tumour, though occasionally it becomes intimately adherent to the newly-formed fatty structures, and superficial ulceration may even then take place in the cutis, which then becomes atrophied. This ulceration, which can generally be brought about by external irritation, seldom goes very deep, although portions of the fatty tissue may nevertheless become gangrenous. Under these circumstances, the ulcers almost always show ill-developed granulations, and secrete a serous stinking discharge. Combinations of lipoma with soft fibroma, with myxomatous sarcoma, and with lymphoma, may and do occur, though very rarely. I have several times observed considerable cavernous dilatation of the veins in lipomata. A disposition to the formation of fatty tumours most frequently exists at the time of life when there is a tendency to develop fat generally, that is, between the thirtieth and fiftieth years. Children very rarely get fatty tumours, though they may occur congenitally on the back, neck, and face; also on the toes, with a simultaneous hypertrophy of the bones (giant growths): they do not generally continue to grow after birth. As a rule, lipoma develops singly, and grows very slowly; it may reach a certain size, and then, in advanced age, cease to grow. The development of multiple lipomata may, however, occur in the subcutaneous cellular tissue: cases have been seen where fifty and even more small tumours have formed simultaneously; they then cease to grow. Multiple lipomata are often mixed

growths. Simple lipoma is *never* infectious, and recurrences never occur after extirpation of fatty tumours.

Friction and pressure have sometimes been observed as causes, leading to the development of fatty tumours; and there likewise exists a certain degree of hereditary tendency.

The diagnosis is in most cases quite easy. The consistence, the lobulated structure which is felt on examining them, sometimes a very definite crackling, which may be appreciated on squeezing certain lobules, are the objective signs of a lipoma. Then the tumours are movable, their slow growth, the age of the patient, and, above all, the region of the body affected, are so many additional aids to diagnosis. They may possibly be mistaken for soft fibrous tissues, or for sarcomata, or for caverno-fatty blood-tumours.

Treatment consists in removing them with the knife. Healing only takes place after an abundant necrosis of gangrenous tissue; in case of large tumours it is usual to remove a portion of the cutis connected with the growth. Erysipelas is especially liable to occur after the removal of fatty tumours, especially when one has to operate on very fat individuals. Nevertheless, the extirpation of the largest lipomata can be safely accomplished, because, for the most part, we have to deal with otherwise quite healthy persons. The extirpation of diffuse fatty tumours is much more awkward than that of circumscribed ones; the general and local reaction is generally much more intense, and yet I have performed several such operations with the best results.

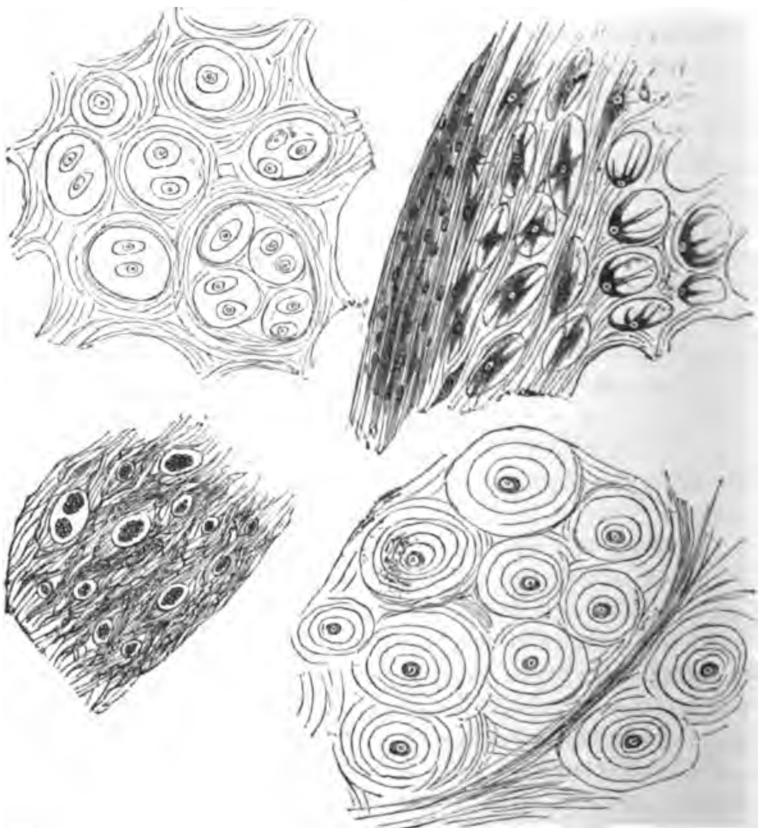
3. *Chondromata—Cartilaginous Tumours*

are tumours which consist of cartilage, either hyalin or fibro-cartilage.

The microscopic elements of pathologically-formed cartilage can assume different forms: sometimes one sees very beautiful round cartilage-cells, such as are found especially in the embryo, and also in a less degree in joint and rib cartilages. Such a complete transition of the hyalin intercellular substance into a homogeneous mass as obtains in normal cartilage, is somewhat rare in chondromata: the intercellular substance belonging to the different groups of cells is differentiated and the hyalin substance forms into fine fibres between the larger group of cells. The latter fact explains why cartilage tumours on section present the appearance of being traversed by interlacing and intercommunicating strands of connec-

tive tissue, which to the naked eye appear like network; the bluish or yellowish shining cartilage is seen embedded between these connective-tissue bundles. Besides this, the tissue of a chondroma differs from that of normal cartilage in being supplied with blood-vessels (which run along these fibrous bands), while normal cartilage is known not to have vessels. The microscopic characters of chondroma present also some further differences from those of normal cartilage. It not infrequently happens that the intercellular substance, whether hyalin or slightly fibrillated, instead of having the uniformly firm consistence of normal cartilage, becomes more gelatinous or friable. Calcification of cartilage, also true ossification,

FIG. 130.



Unusual forms of cartilage tissue from a human chondroma, and from a dog. Magnified 350 times.

are rather common in chondromatous tumours; the form of the cells may be very different (Fig. 130).

As regards the external form of chondroma, they are generally roundish, nodular, and sharply defined tumours which sometimes grow to the size of a man's head. Their growth at first is purely central. In its further course, however, the tumour enlarges partly by the occurrence of new disease foci in the immediate neighbourhood, partly by the conversion of the nearest tissue into cartilage (local infection). Among the pathological metamorphoses, pulpy and mucous softening, and the ossification of some parts, have already been mentioned; as the result of the former process mucous cysts develop in these tumours, in consequence of which the otherwise hard chondromata may present partial fluctuation. It is conceivable that, after complete ossification of the chondroma has taken place, the tumour might cease to grow; and this has actually been observed in a few cases. Superficial ulceration may occur in large chondromata, especially when the skin is highly tense, as the result of traumatic irritation; it is of no great importance. Ulcerative softening in the interior, with perforation outwards, are rare events. I have seen one such case; it was a well-formed typical chondroma, as large as an apple, situated on the sheath of one of the tendons of the foot. Virchow calls the ossifying layer between the periosteum and growing bone osteoid cartilage: hence he christens periosteal and ossifying tumours, which have a structure similar to this osteoid-cartilage, "osteoid-chondroma." I am a little doubtful how we are to distinguish such tumours, and I have examined a good many, from periosteal ossifying round or spindle-celled sarcomas; hence I would prefer not to separate Virchow's osteoid-chondroma from sarcomas.

Occurrence.—Cartilaginous tumours occur very frequently on the bones. The phalanges and metacarpal bones are the most frequent seat of chondromata; more rarely the analogous bones of the foot. On the other hand, the chondromata are almost always multiple, sometimes in such numbers that scarcely a finger is free; next come the thigh bone and the pelvis as favourite seats of chondroma. In these positions they attain their largest size, and may lead to the most complete destruction of these bones. More rarely chondromata may occur on the bones of the face, very occasionally also on the skull; a little more frequently again on the ribs, and on the scapula. They occasionally, though rarely, develop in the sheaths

of tendons. In soft parts, and especially in glands (testes, ovaries, mamma, salivary glands), these tumours have also been observed;

FIG. 131.

Chondroma of the fingers.

sometimes they are completely developed chondromata, sometimes only single bits of cartilage along with much larger developments of sarcomatous or carcinomatous growth.

The development of chondroma is chiefly peculiar to youth, not that it occurs exactly in young children, but about the age of or just before puberty; most chondromata date from this period, even although they may not be observed until in later life. These tumours sometimes occur after injuries; they grow exceedingly slowly, and appear from time to time to come to a complete stand-still. I have known patients tell me that they believe that their tumours have remained unchanged for years, and that merely accidental circumstances have induced them to wish them removed. Sometimes, however, they grow more rapidly and become infectious. Cases are on record in which finally cartilaginous tumours have occurred in the lungs (embolic), and caused death. O. Weber has also observed hereditary tendencies to the chondroma diathesis. In the combinations, already referred to, of cartilage tumours with sarcoma or carcinoma, the former exercises no influence on the prognostic characters of the tumours as a whole.

The diagnosis and prognosis may readily be inferred from what has been said. I need only add that the softer and cystoid forms of chondroma are mentioned in older works under the names of colloid ulcers, gelatinous cancer, alveolar cancer &c. As the epithelial elements, and also the connective tissue framework, may become gelatinous (mucoid, colloid, myxomatoid), in fibroma, chondroma sarcoma, as well as in adenoma and glandular cancer, we must always observe veіy carefully what we have before us; we

shall often enough be in doubt as to the significance of the histological elements and their metamorphoses, and also as regards the name we ought to choose for the disease.

As concerns treatment: this must consist in the removal of the tumours, provided that it can be done without danger to life. The, as a rule, very large chondromata of the pelvis must be left alone; tumours of the thigh, which also have often grown to a very large size before the patient comes under our care, can only be got rid of by disarticulating the thigh, for which there will scarcely be the necessary indications, until the leg, in consequence of spontaneous fracture of the femur from trophic disturbance in the bone itself, has become quite useless. Chondromata of the fingers most frequently come to operation, not because they are painful, for they are generally quite painless, but because they interfere with the action of the fingers; it is true that this only occurs very slowly and gradually, by which time the tumours have attained a considerable size. So long as these patients can use their misshapen fingers, they seldom want an operation performed; nor should we be justified in insisting on it. As regards the nature of the operation: in many cases in which the tumours, although firmly adherent to the bone, are placed somewhat sideways, one would try to remove the tumour by dividing the skin and carefully pushing it and the sheath of the tendon on to one side, and then cutting it off either with a knife or a saw. This, however, is only possible in a certain number of cases, if we wish to remove the whole tumour, which, of course, is necessary. The mass of cartilage very frequently invades the whole medullary cavity of the bone, and besides this, very severe inflammation of the sheaths of the tendons may supervene after such operations. Dieffenbach was of opinion that any remnants of the chondroma subsequently ossified and remained stationary; but concerning this point there are not sufficient well-observed cases to substantiate it. For these reasons the removal of chondromata from bones is only possible in a few instances, and should be confined to cases where the tumours are still quite small. The operations may be successful nevertheless. In two cases, where I succeeded, there has as yet been no recurrence. If the tumours have already acquired a considerable size, we should postpone the exarticulation of the fingers until the hand has become absolutely useless.

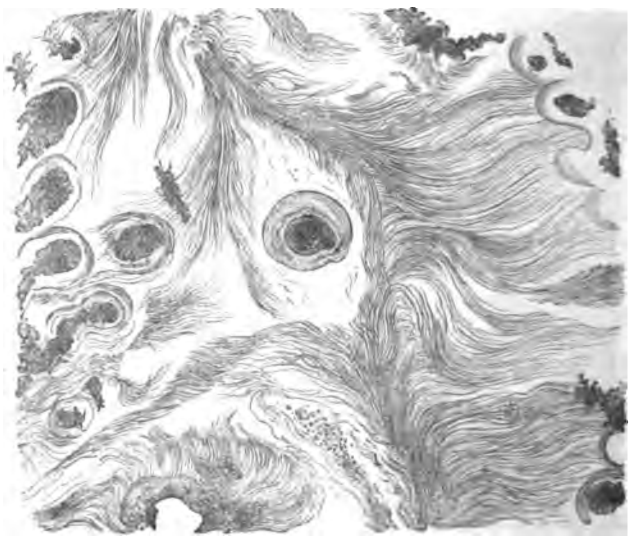
4. *Osteoma. Exostoses.*

We designate, under these terms, abnormally developed masses of bone which, in a circumscribed form, constitute a tumour; they have an independent growth, and are not dependent on chronic inflammation. The formation of bone occurs also occasionally in many other forms of tumours, especially in such as form in bone, as we have already pointed out in chondromata. The term osteoma is, however, limited to tumours which consist entirely of bone. I will here just mention that not only new formations of entire, though not very regularly formed, teeth occur in ovarian cysts, and in the antrum of Highmore, but also that outgrowths from the

FIG. 132.

Odontoma of a molar tooth. Natural size.

FIG. 133.



Section from an Odontoma. Magnified 100 times.

teeth themselves, and consisting of real ivory—ivory exostoses (odontoma, from ὀδούς, tooth, Virchow), have been observed. But they are very great rarities, and must be regarded merely as curiosities. As regards the anatomical structure of osteomata, they are found to consist partly of spongy bone substance, filled with ordinary bone marrow, and partly of an ivory-like substance, arranged in regular lamellæ, analogous to the cortical substance of the ordinary long bones. Hence we may distinguish spongy osteomata and ivory osteomata. A third variety of osteomata is formed by the ossification of tendons, fasciæ, and muscles; but their right to be classed as tumours at all is certainly very doubtful.

(*a*) *Spongy osteomata, with cartilaginous capsule* (exostosis cartilaginea). These tumours occur almost exclusively on the epiphyses of the long bones. They are outgrowths from the epiphyseal cartilage, on which account Virchow has very appropriately called them "ecchondrosis ossificans" (see Fig. 134). On their rounded, nodular

FIG. 134

A pedunculated spongy osteoma at the lower end of the femur. After Péan.

surface there is generally found a covering, about a line or a line and a half in thickness, of beautifully formed hyalin cartilage, which apparently grows partly in itself and partly peripherally from the periosteum, or rather from the perichondrium, and then rapidly ossifies towards the centre. The newly formed mass of bone is, from its very commencement, most intimately connected with the spongy substance of the epiphyses, so that the hard tumour rests immovably on the bone. It is the very nature of these

osteomata that they should occur only in young subjects. Tibia, fibula, and humerus are, according to my observations, their most frequent seats.

(*b*) *Ivory osteomata.*—They consist of compact bone-substance with Haversian canals and lamellæ; they develop on the bones of the face and of the skull (see Figs. 135 and 136), and on the pelvis, on the scapula, on the great toe, &c., forming roundish, slightly roughened, or sometimes smooth tumours.

A third variety of abnormal tumour-like formation of bone is the ossification of tendons, of fasciæ, and of muscles, which occurs, as a rule, in a series of tendons and fasciæ after previous shrinking. Thus, the skeleton in such cases—generally young subjects—is covered with as many as twenty to fifty long, sharp bony processes, at points where tendons are usually inserted (see Fig. 137). Sometimes the ossification occurs primarily in the fasciæ of muscles, as I once had an opportunity of seeing in Zürich. There are cases on record in which this ossification has gone on to such a degree that all the muscles of the shoulder and of the arm have ossified, and rendered any movement of the upper extremity altogether impossible. This kind of bony growth, as also the so-called "drill-bones," are to be regarded as the product of a chronic inflammatory process just in the same way that a production of true bone may occur abnormally in the cranial or within the spinal dura matral sheaths. By "drill-bones" we mean the development of masses of bone in the deltoid muscle, and especially at that point where the rifle strikes during drilling. These growths, however, only occur in a very few soldiers, and one must always pre-suppose some peculiar predisposition to their formation. The ossification of tendons which occasionally occurs, especially at their point of insertion into bones, is also a very remarkable occurrence, and reminds one of what occurs normally in birds.

The predisposition to the formation of osteoma is closely allied to that of chondroma; it also occurs especially in young subjects, and more frequently in men than in women, while infancy is almost completely free. As regards epiphyseal osteomata, which we might just as aptly call ossifying chondromata, it is in the very nature of things that they should not occur beyond about the twenty-fourth year. Other osteomata are also found, for the most part, previous to the thirtieth year. Observations on this point are not very numerous, as the disease is far from common. The fact that

osteomata occur by preference in young people is remarkable, and stands in strong contrast with the rule that ossification occurs especially in advanced age.

The costal and laryngeal cartilages, sometimes also the vertebral ligaments, ossify in advanced life, and calcification of the arteries also belongs to an almost natural, senile marasmus; nevertheless, the formation of bony tumours but very rarely takes place, and even though these tumours may be found in old people, they are generally known to have existed since youth. Osteomata are just as often multiple as single. As a rule their growth is exceedingly slow, and is often arrested as age advances. The epiphyseal exostoses cease to grow when the skeleton attains its full size, and then the spongy bony substance becomes more compact. It is only in very rare instances that the ossification of tendons and muscles proceeds to such a degree that the movements become completely impeded. In a few cases development of bone has occurred in the lungs. The inconveniences caused by osteomata are not, as a rule, very serious; they give rise to no pain, and they are not

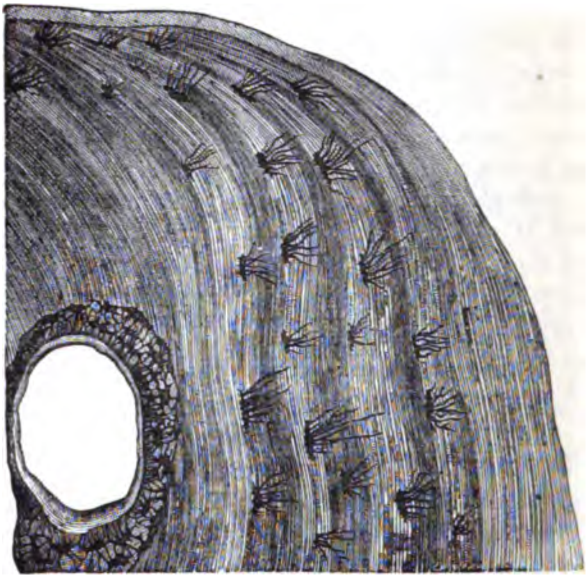
Fig. 135.

Ivory osteoma of the skull.

tender when handled. Osteomata which are situated in the neighbourhood of a joint frequently interfere with its functions. Tumours of this kind occurring on the bones of the face give rise to unpleasant disfigurements; exostoses of the great toes may

prevent the boots being pulled on; ossification of tendons and of muscles limit their movements, and sometimes arrest them

FIG. 136.



A thin section of an ivory osteoma of the skull.

altogether. Unfortunately, in the latter case, on account of the great extent, operative measures are the least indicated, the more so as the disposition to further deposits would still continue. As regards the operation for exostoses, it consists in sawing off, or chiselling off, the tumour from the affected bone. As they sometimes occur in the neighbourhood of a joint, as we have already said, the joint may occasionally get opened at the operation; therefore it is both *unnecessary and unwise* to undertake such operations, except when the function of a joint is so seriously interfered with as to justify an operation which is dangerous both to life and limb. We shall be the less inclined for such operations without special indications, seeing that, in time, mere tumours cease to grow. On epiphyseal exostoses we sometimes find mucous bursæ, containing adherent or loose ossifying cartilages; the bursæ generally communicate with the joint in the neighbourhood of which they grow. According to the researches of Rindfleisch, these mucous bursæ are

enormously distended prolongations of the articular synovial membranes. I once allowed myself to be persuaded by a patient to

Fig. 137.

Osteomata at muscular attachments. After O. Weber.

remove one of these exostoses at the lower extremity of the femur, and to extirpate the abnormal synovial sac at the same time; the patient died of septicæmia. In another case the synovial bursa of an exostosis opened spontaneously with some symptoms of inflammation; suppuration of the elbow followed, which ended in anchylosis.

LECTURE XLVI.

5. Myomata. 6. Neuromata. 7. Angiomata—A. *plexiform*; B. *cavernous. Methods of operating.*

5. *Myomata.*

WHETHER myomata consisting entirely of transversely striated muscular fibres or muscular fibre-cells really exist must for the present remain undecided. I am not aware of any such case. The occurrence of newly-formed striped muscular fibres is exceedingly rare in tumours, and a tumour consisting entirely of them has never been found; they generally occur, as an accidental circumstance, in sarcoma or carcinoma (of the testis, or ovary, or mamma), or in very complex and compound tumours. I have examined tumours in which there were grades of development of the muscular fibres, but the propriety of calling such tumours "myomata" has been disputed. I feel unwilling to say anything against this, because we may not call tumours which merely consist of developmental stages of connective tissue fibromata, and because I have already raised doubts as to the propriety of calling uterine fibroids, which consist of spindle-cells, "myomata," so long as we are not quite clear on the relation of spindle to muscle cells. In the prostates of elderly people newly-formed unstriped muscular fibres occur in large quantities, which occur sometimes in the form of single nodules, sometimes as a diffuse enlargement of the entire organ. There is certainly no great objection in calling this so-called hypertrophy of the prostate (a certain amount of glandular enlargement is usually associated with it). Similar myomatous nodules are found in the tunica muscularis of the œsophagus and stomach. A short time since I successfully extirpated a pedunculated myoma from the bladder of a little boy; it seemed to spring from the tunica muscularis of the bladder. Clinically, nothing definite can be said of myomata under these circumstances. The tumours, which I con-

sidered as young myomata in muscle, were of a medullary appearance on section, fascicular, with an irrepressible tendency to local recurrence, and led in that manner to death.

6. *Neuromata.*

It has already been mentioned that the term "neuroma" is used for all tumours which grow on nerves. This is, if you will, a practical misuse, which it will be difficult, nevertheless, to root out. By true neuroma we mean a tumour which consists entirely of nerve-fibres, and especially of those with double contours. These formations only seem to occur on nerves, and they are extremely rare. The neuromata on amputation-stumps have already been referred to; whether they are real neuromata is warmly disputed. True neuromata are always very painful. Many fibromata on and in nerve-trunks contain very peculiar fine bundle-like filaments richly supplied with nuclei, which are often and well called grey non-medullated fibres, as Virchow considers them—a view which will give true neuromata a wide signification, and divide them into medullated and non-medullated varieties. I could not trust myself always to distinguish a non-medullated nerve from a fibroma in nerves, and I would not require others to do it. Tumours composed of spindle-cells arranged in bundles are probably more frequently myomata and neuromata than young fibromata, though the proof of one or the other would be difficult to obtain. They tend to be multiple and to regional recurrence, and hence the prognosis must always be cautious. It is seldom possible to dissect a neuroma away from a nerve trunk; generally a piece of the latter must be cut away with it.

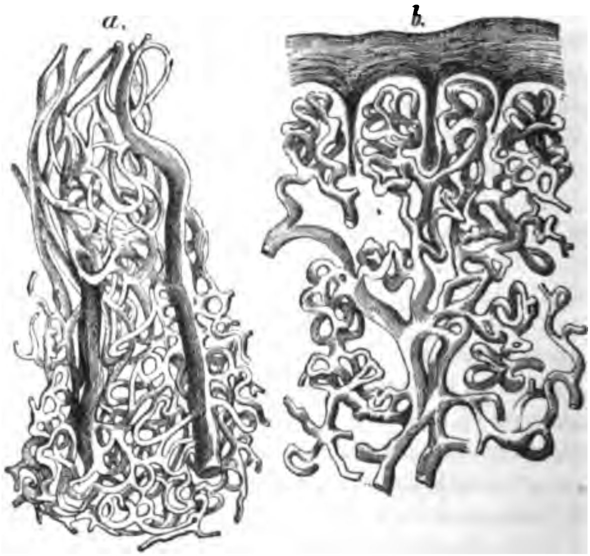
7. *Angioma. Vascular Tumour.*

By these terms we understand tumours which are composed either entirely or in part of vessels which are held together by a small quantity of connective tissue. They have also been called "erectile tumours," because, as they are more or less filled with blood, so are they firmer or softer, larger or smaller. The ordinary form of varicose swelling of the veins and aneurism of individual arterial stems are excluded from this category. Cirsoid aneurism might, however, be included, as also certain forms of aneurismal varix; but, as this is not usual, we have discussed these diseases earlier on.

We have two different forms of vascular tumours to consider.

A. *Plexiform angioma*, or telangiektasis (from τέλος, ἀγγεῖον, ἔκτασις). It is the commonest variety. This new growth is entirely composed of dilated and enormously hypertrophied and tortuous capillaries and intermediate vessels; accordingly, as the hypertrophy of the vessels or purely ectasis predominates, so it appears either as a tumour or as a red patch in the skin. The plexiform angiomata, which we are about presently to describe, occur almost exclusively in the skin. They are sometimes of a dark cherry red, sometimes of a steel-blue colour; sometimes they are no larger than a pin's head, and sometimes as large as a small plate; some are rather thick, others scarcely project above the level of the skin. There are rare varieties, in which it is not merely a red spot or a small tumour, but an extensive and diffuse redness, extended over a considerable area of the surface of the body, and in which, even to the naked eye, the distended and tortuous finer capillaries can be distinguished on the surface of the cutis, shining through the epidermis.

FIG. 138.



Conglomeration of vessels from a plexiform angioma. Magnified 60.
a. Hypertrophied vascular loop around a sweat duct (which is left out in order not to complicate the drawing). *b*. Hypertrophied vascular loop in the papilla of the mucous membrane of the mouth.

The anatomical examination of a small extirpated angioma of this kind shows it to consist of small lobules, from the size of a hemp-seed to that of a pea, and on examining with a microscope a section which has been previously injected artificially, it is seen that this lobulated condition is due to the peculiar arrangement of the capillaries around the sweat-ducts, hair-follicles, fat-glands, and fat-lobules, all of which are separately diseased. The colour—sometimes blue-red, sometimes pale blue—of these tumours is dependent on this circumstance, that in the first cases the capillaries of the uppermost cutis layer, and in the second the deeper vessels are affected. As a rule, this capillary hypertrophy does not pass beyond the subcutaneous cellular tissue; only in rare cases does it invade deeper structures, as, for instance, muscles, from which we conclude that these new growths proliferate not from the centre, but especially from the periphery, and destroy the affected part.

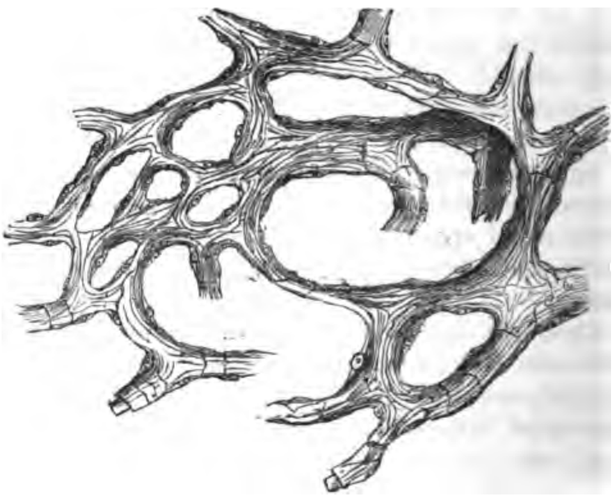
Most of these tumours can be emptied by pressure, though somewhat slowly, and they may refill again as soon as the pressure is moved. Nevertheless, there are small telangiektases in which, besides the vascular hypertrophy, there is a proliferation of the connective and adipose tissues, so that on pressure they cannot be entirely emptied. When these new growths are on the surface of the cutis, and when, after extirpation, the blood has emptied itself, scarcely anything abnormal can be detected with the naked eye; on further section such a tumour would present a pale reddish, soft, small lobulated substance, in which no vessels could be seen with the naked eye, simply because the disease is confined to the capillaries and intermediate vessels, and one or two of the smaller arteries.

b. Cavernous angioma, or cavernous tumours of veins.—I will first describe their anatomical characters in order that you may at once rightly appreciate how they differ from plexiform angioma. An extirpated cavernous angioma is quite obvious to the eye on section, and is recognised by its great likeness to the corpus cavernosum penis. One sees a whitish, tough network, which appears empty or partly empty, or filled in places with a red or colourless clot, perhaps with small round, chalky concretions, so-called phleboliths. We must imagine this network filled full of blood previous to the extirpation. The limitation of this cavernous tissue, which can form in all tissues of the body, is by a kind of capsule, which is very definite; in some cases, however, this limitation is

less definite, and gradually merges, sometimes here, sometimes there, into surrounding structures.

The microscopic examination of this network, which is sometimes made up of only thin threads, sometimes of membranous capsules, shows that the strands consist of the remains of that tissue in which the cavernous ectasis has commenced. The interior wall of these blood-filled spaces is lined in the majority of cases with a flattened, continuous layer of endothelial cells, which along the borders of the strands assume a spindle-shaped outline; and even these anatomical conditions speak in favour of our having to do more especially with dilated veins. The manner in which this peculiar structure comes to be formed has been variously explained. If we possessed any definite ideas on the development of the corpus cavernosum penis, we might be able to draw some conclusions therefrom, on account of the very great resemblance of these two structures. The three chief hypotheses which are at present held on the development of cavernous tumours are the following:

FIG. 139.



Trabecular network from a cavernous angioma of the lip (the blood must be imagined as filling up these interstices). Magnified 350.

(1.) It is assumed that the cavernous spaces, at first, develop out of connective tissue, and then secondarily get into communication with the vessels; in consequence it has even been thought that blood might be formed direct from connective-tissue cells outside

the vascular system; the partitions of this network multiply by inherent growth, by bud-like or diverticular outgrowths of the connective tissue (Rokitansky). The hypothesis of the formation of blood outside the circulation has much against it, although the most recent researches, by A. v. Winiwarter, on cavernous lymphangiomata, support the view that in the immediate neighbourhood of the vessels, perhaps even in their very walls, masses of cells collect, which soften in their interior, and then open up communications with the blood-vessels around them. We shall presently become acquainted with similar processes when speaking of villous sarcomata. (2.) We assume that there are found small dilatations of venous vessels, one close to the other, the walls of which, gradually thinning away and finally disappearing at those places where they come in contact, are absorbed. In favour of this theory is the fact, that a similar and gradual dilatation of the veins may be very clearly followed both on the cutis and in bone during the development of these tumours. (3.) Rindfleisch strongly insists that the ectasis of the vessels, especially in cavernous tumours which form in the orbital fat, is always preceded by a small cell infiltration of the tissue, and that this is followed by a kind of contraction of the tissue, and a consequent stretching of the vessels, the lumina of which, by continued shrinking of the intervening tissue, must consequently become larger.

I have long held, for many reasons, that a process very similar to inflammation must go on both in the plexiform and in the cavernous angioma, for neither the latter (which is hardly applicable for cavernous tumours of bone) nor the two former hypotheses appear to me to be sufficient to explain the causes of, and peculiar differences in, the vascular distribution. There is still a difference, which cavernous tumours present, to be mentioned: they are either attached to the larger vein-trunks, for instance, the subcutaneous veins, like a pouch, or a number of small vessels, arteries, and veins, supply the capsule of the cavernous tumour. Finally, we must mention that cavernous venous ectasia may occur accidentally in other tumours, as, for instance, in fibromata and lipomata, as has already been mentioned. Some years ago I extirpated a lobulated lipoma, which had developed beneath the scapula of a young healthy man; the central lobules had all degenerated into a cavernous mass. Cavernous angiomata develop especially often

in the subcutaneous cellular tissue, less frequently in the skin and in muscles, very rarely in bones; fairly often in the liver, on its surface, sometimes also in the spleen and in the kidneys. In some instances they are very painful, in others quite painless. The diagnosis of cavernous angiomata is not always easy; when they occur in the skin they may always be confounded with more deeply-seated telangiektases, although the blood may be squeezed out of a cavernous vein-tumour more easily than out of a telangiectasis. The deeply-seated tumours of this kind are always difficult to diagnose with any certainty. They generally present marked fluctuation, are rather compressible, and swell out in prolonged respiration; the last two symptoms are, however, not always very definite, and hence they may be mistaken for lipoma, cysts, and other soft tumours; indeed, in some cases, the mistake cannot be avoided.

About one half of the angiomata are congenital, or at least they are developed very shortly after birth. If they develop during life, it is usually during infancy or childhood, and it is quite exceptional for vascular tumours to develop during manhood or old age—a circumstance which is decidedly striking when we remember that the disposition to aneurism and arterial diseases is so common at this time of life. The small intermediate vessels and capillaries at certain places also show through the skin appreciable dilatations, and we may observe on the face of a bushy, healthy old man ruddy cheeks, as in youth; but yet it is not the same uniform rosy red of a young girl, but a bluish red, and when we regard it more closely we find that they are numbers of very tortuous vessels; in cases this redness comes in patches. Nevertheless, these small vascular ectasia do not occur in all old people, and there we must assume that there is a predisposition in some persons. However, as we have said, although advanced age is more disposed to vascular disease than any other time of life, yet blood tumours proper only develop during youth. That telangiectases, which in popular phraseology are often spoken of as "mothers' marks," are hereditary, is established beyond a doubt. A number of tales and legends, in which a lost child has been recognised by a "mark" inherited either from its father or its mother, all point in this direction. We might doubtless learn still more about the inheritance of vascular tumours if we were to consider the subiect of the hereditary nature of vascular disease and

vascular changes generally. And although plexiform and cavernous angiomata must be considered as anatomically different, and also different from the various form of varices and aneurysms, it is, nevertheless, clear that a predisposition to dilatation of vessels exists in all these cases. This, it seems to me, is most probably hereditary, and the above-named diseases ought therefore only to be considered as apparent varieties of such a predisposition peculiar to various periods of life. Our attention has been so exclusively directed to the anatomical conditions of these tumours individually that we have paid too little attention to these allied diseases as a group.

As regards the further history, we may say that telangiectases may occur either singly or multiple. Their growth is always slow, painless, and takes place mostly on the surface, though sometimes chiefly towards deeper structures, and generally at the expenses of the tissue in which the disease starts. Their growth occasionally becomes arrested after the course of years without doubt, but the tumours remain unchanged. In other cases their growth proceeds uninterruptedly, so that the tumours, as I once saw in a child five years old, may attain the size of a man's fist.

It is common to find two or three telangiectases together, especially on the hairy scalp at birth, or they may develop very shortly after birth; sometimes their number may be six or eight. I have seen two cases of flat, congenital plexiform angioma of the left half of the face, which, partly as the result of ulceration, and partly from unknown causes, healed up; that is, there appeared here and there white cicatricial spots, where the vessels were obliterated, while elsewhere in the peripheral portions the tumour continued to grow vigorously. Cavernous angiomata are seldom congenital; they must come on in infancy or childhood, seldom in later life. Their favourite position is, as we have already remarked, in the subcutaneous connective tissue, frequently in the face, rarely on the trunk or on the extremities. They sometimes occur in considerable numbers, and yet so that, as a rule, a particular vascular district can be recognised as a diseased one. Thus an entire arm, or foot, or leg, the whole or a portion only of the face, may be the seat of such a tumour. The symptoms which they give rise to, besides the disfigurement, are a certain amount of weakness in the muscles, and sometimes also pain in the neighbourhood of the diseased part.

The tumours may grow to a considerable size, and on that account, especially when on the head, they may become dangerous, the more so as, by continued growth, they disturb and destroy the bones. From some cases which I have observed I am inclined to think that, in consequence of thrombosis in the cavernous spaces, contraction and atrophy of these tumours may take place (especially in cavernous tumours of the liver); a complete disappearance of angioma from spontaneous obliteration has, however, been observed. The treatment of these cases is very manifold; small congenital vascular tumours may disappear in the course of a few months spontaneously. Operations are undertaken with two different objects in view.

1. *Operations that aim at coagulating the blood with subsequent obliteration and shrinking of the tumour.*—Here we must count injection of liquor ferri sesquichlor. into the tumour, or the passage of threads soaked in liquor ferri; also the application of red-hot needles, or the galvanic cautery and a platinum thread, which, after being put round the tumour, is heated by the galvano-caustic apparatus.

2. *Operations by which the tumours are completely removed.*

(*a*) By ligature, which in the case of broad-based telangiectases must be double or multiple. A needle, armed with a double thread, is passed beneath the tumour, and one of the threads is tied tightly round the base of the tumour in one direction, and the other thread around the base in the opposite direction.

(*b*) By vaccinating on the tumour, in order that by the dropping off of the scab the diseased tissue may be eliminated.

(*c*) By cauterisation; for which purpose the best means is fuming nitric acid; it can be applied with a little stick as large as the handle of a penholder until the surface of the angioma becomes changed to a marked yellow colour. Concentrated sulphuric acid will accomplish the same result.

(*d*) By extirpating with the knife or scissors.

After a little operative experience the choice of a method for the individual cases is not difficult. For superficial angiomata, if they are not too extensive, and situated at a part where the after cicatricial retraction will not produce any considerable disfigurement, as in many parts of the face, I consider cauterisation with fuming nitric as the normal proceeding. For medium-sized plexiform and cavernous angiomata extirpation with knife or scissors is the

surest operation. To obviate any considerable hæmorrhage at such an operation, compression of the surrounding tissue by a clever assistant must be resorted to, or else the bleeding may be arrested by means of a rapidly applied ligature around the peripheral border of the tumour. For many angiomata of the face excision is to be preferred to cauterisation, because the incisions may be so made that contractions of the mouth or eyelids do not take place to any appreciable extent. On the other hand, there are cases in which extirpation is altogether impracticable either on account of the size, or the seat, or the multiplicity of the tumours. I treated a child with a still growing cavernous tumour, which extended down from the bridge of the nose, involving the nose and the whole thickness of the upper lip. If one had desired to extirpate this, it would have been necessary to have removed the whole nose and the upper lip; this, of course, was not to be thought of, and I, therefore, tried cauterisation with hot needles. The treatment had gone on for three months, and ought to have continued for another three months, although the greater number of the cavernous spaces were already obliterated; but the mother of the child lost patience, and I have not seen it since. I have had remarkably good results during the past years from making deep punctures into these diffuse angiomata with the galvano-cautery and with the ordinary setaceum candens. In addition to partial destruction of the new growth there is doubtless also atrophy; further, small portions may, here and there, be excised. I prefer this kind of cauterisation to the injection of liquor ferri perchlor., because in a few cases extensive sloughing and gangrene have taken place, and because injecting is sometimes interfered with by the formation of coagula within the fine canulas. I have not much experience of the use of threads soaked in liquor ferri (Roser). While some surgeons have secured very vigorous reaction, in the cases where I tried this treatment there was but very slight inflammation, although I left the threads *in situ* for fourteen days; contraction then slowly occurred; I am not yet sure whether this plan prevents recurrence. The other methods which I have mentioned are altogether of secondary importance. Vaccination very frequently does not penetrate sufficiently deep, and the ligature is a tedious and not always reliable plan, and it is not entirely free from the danger of secondary hæmorrhage.

I may append also the following:

(1) Cavernous lymph tumours (lymphangioma cavernosum), a

very rare form of new growth, which anatomically is constructed on exactly the same plan as the cavernous blood-tumours, but with this difference that, instead of blood, there is lymph in the meshes. This kind of tumour occurs congenitally in the tongue, as a form of macroglossia (there is also a fibrous form of macroglossia), and on the neck as a kind of cystic hygroma. I have seen both occur in the same child. I have also seen this kind of tumour in young subjects at different other parts of the subcutaneous cellular tissue (lip, cheek, chin). That varices of the lymphatic vessels in the thigh may become cavernous lymph-angiomata has already been mentioned, as also the most recent researches on the development of cavernous lymph-angiomata by A. v. Winiwarter.

(2) Nævus vasculosus, the so-called mother's mark. This is a plexiform angioma of the most superficial vessels of the cutis; they, however, cease to grow from the moment of birth. There is no other difference between a growing angioma and a "mother's mark" than this. I have already mentioned that hypertrophy of the skin, pigmentation, vascular ectases, and the formation of hair may be curiously combined in the congenital marks. If they exist on the face, and are not too extensive (they sometimes involve the whole half of the face), a total or a partial extirpation, with subsequent plastic operation, may be undertaken; or, according to circumstances, cauterisation may be preferable. Many of these marks, in which usually only the tips of the papillæ are affected, may be considerably improved, and even eventually cured by removing a very superficial layer of the cutis.

LECTURE XLVII.

8. *Sarcoma. Anatomy.* A. *Granulation sarcoma;* B. *Spindle-cell sarcoma;* C. *Giant-cell sarcoma;* D. *Reticular sarcoma;* E. *Alveolar sarcoma;* F. *Pigmented sarcoma;* G. *Villous sarcoma. Tubercle. Psammoma;* H. *Plexiform (cancroid, adenoid) sarcoma. Cylindroma. Clinical characters. Diagnosis. Course. Prognosis. Mode of infection. Topography of sarcoma. Central osteosarcoma. Periosteal sarcoma. Sarcoma of the breast; of the salivary glands.* 9. *Lymphoma. Anatomy. Relations to leucæmia. Treatment.*

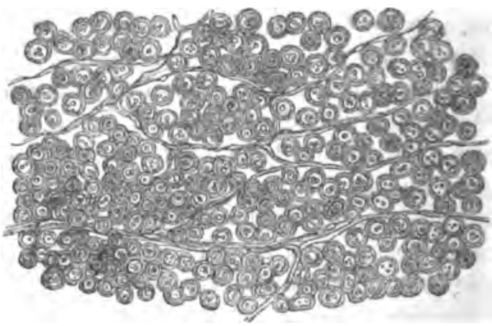
8. *Sarcoma.*

Over no group of tumours has there been so much, or so long, uncertainty as to the anatomical definition and limitation, as in sarcomata. The comparatively old definition (from σάρξ, flesh) signified nothing more than that these tumours, when cut into, had a flesh-like appearance. According to this, it was naturally impossible to make a diagnosis, for it was rather a matter of uncertainty what should be called flesh. The attempt to reserve the name sarcoma only for tumours which consisted of muscle (Schuh), and to identify it with what we now call myoma, found but little acceptance. Subsequently the anatomical signification of sarcoma became somewhat more definite, as it came to include all tumours rich in cells, which presented no definite alveolar structure, and which were not carcinomatous. It is, however, only within the last ten years that the following histological definition has received general acceptance, and come to be at all definitely applied. *A sarcoma is a tumour consisting of tissue which belongs to the developmental series of connective tissues* (connective tissue, cartilage, bones, muscles, and nerves), and *which do not, as a rule, go on to the formation of a finished type of tissue, but tend rather to peculiar degenerations of the developmental forms.* Many pathologists would

gladly exclude "muscles and nerves" from this definition, but when speaking of spindle-cell sarcoma I will give my reasons why I cannot sanction this. If one would include inflammatory neoplasia in their different stages as instances of sarcoma (Rindfleisch), I can assent, because this view would be fairly well included within my definition. That the cellular elements of the vessels may also serve as matrix for the formation of sarcoma is no longer doubtful, and has been especially insisted on in the recent observations of Köster, Tillmanns, Arndt, and others. It seems to me, however, rather early to hazard the opinion that all sarcomata have a similar origin. In some sarcomata contractile cells have been found (Lücke, Grawitz), but still most researches of this kind have resulted negatively, and hence the observations cannot be further utilised at present.

After this anatomical basis for the definition of sarcomata had been made, it was soon discovered that sarcomata might be diagnosed by the naked eye, and that something also might be said clinically of the peculiar course of these tumours. I am rather of opinion that at present the subdivisions, made according to the histological peculiarities of the various sarcomata, are of no great value in the diagnosis of sarcoma during life, and that the diagnosis, prognosis, and course of these tumours depend so much on the place at which they occur, and on the rate at which they grow, that I prefer to sum up my clinical remarks on sarcomata later on, and here simply and chiefly to consider their histology. We shall distinguish the following varieties of sarcoma.

FIG. 140.

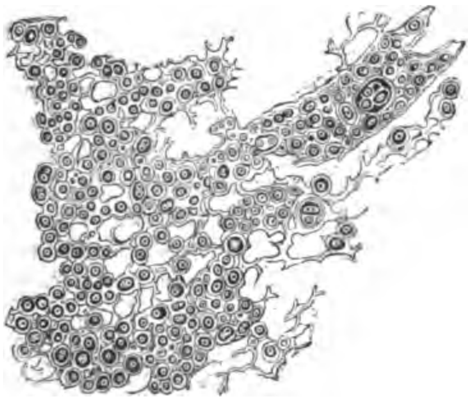


Granulation sarcoma. Magnified 400 times.

(A) *Granulation sarcoma. Round-cell sarcoma* (Virchow).—This tissue is like or is very similar to the uppermost layer of granulations;

it always contains a preponderance of small round cells, like lymph-corpuscles; the intercellular substance is sometimes scarcely perceptible, while at others it is abundant; it may be completely homogeneous, as in the neuroglia (Virchow's glioma and glio-sarcoma), or it may be delicately striated (Fig. 140), or even fibrous and œdematous (as in large mammary sarcomata). It may be also reticular, and it then comes in close relationship to the tissue of lymphoma (Fig. 141).

FIG. 141.



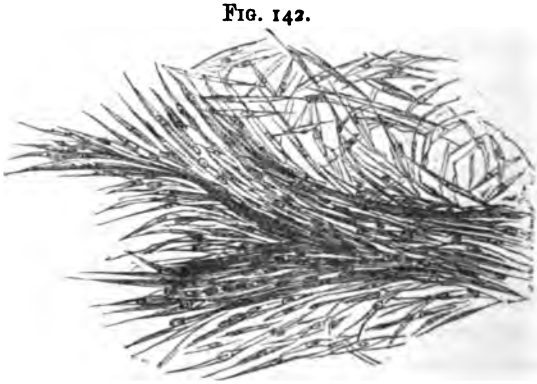
Glio-sarcoma. After Virchow. Magnified 350 times.

(B) *Spindle-cell sarcoma.*—This tissue is formed of closely lying, mostly thin, elongated spindle-cells, so-called fibre-cells, which are usually arranged in bundles. There is seldom any intercellular substance; but it may be present, either white and homogeneous or fibrillated. If there is much fibrous tissue, the tumour is then called fibro-sarcoma or fibroma. Formerly this spindle-cell tissue was always regarded as young connective tissue (tissu fibro-plastique, Lebert); but, as the result of my own histogenetic researches in embryos, I have long protested against this view, because a spindle-cell tissue, similar to that found in these sarcomata, is never present in embryonal connective tissue, not even in tendons.

The physiological prototype of this tissue is young muscular and nerve tissue; according to this these spindle-cell sarcomata would be young myomata or neuromata. Virchow has still further elaborated this same view, especially as regards uterine fibroid tumours. I have protested against this view and its consequences

because the diagnosis is always doubtful in special cases. If a tumour which consists of elongated spindle-cells, the extremities terminating in fine fibres, develops in a nerve, it is very natural to regard such a tumour as a neuroma, the elements of which, at any point, have not come to full development. And if a spindle-cell tumour develops in muscle, and its fibres show a distinct band-like appearance, and even dottings, as if in commencing striation, we could not be blamed for calling these tumours "myomata," in the belief that we were dealing with young muscular tissue that had not got beyond a certain stage of development. Thus far there is no objection to such views. But if a spindle-cell sarcoma occurs on the skin, or on the penis (of which I had a remarkable example a little while since), there may be great difficulty in deciding whether it is a neuroma, myoma, or fibroma; for there are nerves, muscles, and connective tissue both in the skin and in the penis. If, therefore, there be nothing typical either in the arrangement or form of the cells, and if the histological origin cannot be clearly made out, then we must content ourselves with the term "spindle-cell sarcoma" (Fig. 142). In such case we have a fibrous tissue the

FIG. 142.



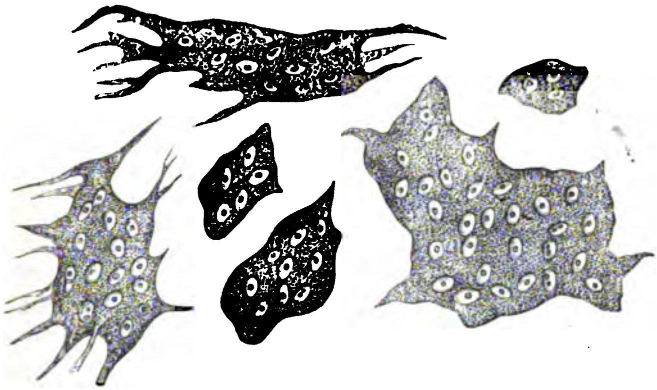
Spindle-cell sarcoma.

development of which has not advanced beyond the production of spindle cells. However, I can state from my own observations that the course and prognosis of these tumours are scarcely dependent on the precise mode in which they originate, but much rather on the locality they occupy, the rapidity with which they grow their consistence, and their other clinical features.

(c) *Giant-cell sarcoma*, according to Virchow, is that variety of sarcoma in which very large cells are found; they are sometimes

round, sometimes irregular in shape, and provided with processes (tails) (Fig. 143).

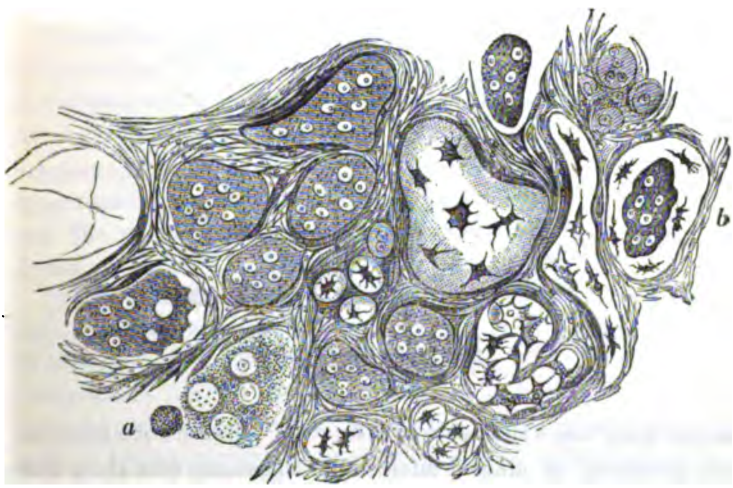
FIG. 143.



Giant cells from a sarcoma of the lower jaw. Magnified 400 times.

These cells, which occur normally in fœtal marrow, though of somewhat less size, have excited great astonishment on account of their size: they are the largest masses of unformed protoplasm with which we are, as yet, familiar in the human subject: they may contain thirty or more nuclei, and their mode of formation from a single cell is, on account of a series of transition forms, easy to follow (Fig. 144).

FIG. 144.



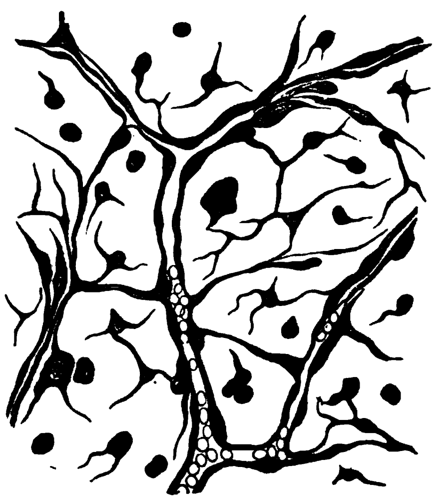
Giant-cell sarcoma, with cysts, and points of ossification from the lower jaw. Magnified 350 diameters.

These giant cells may occur both in spindle-cell or fibro-sarcoma; they may be found also sporadically and smaller in granulation and myxo-sarcomata. They are likewise found in central osteo-sarcomata, and somewhat less frequently also in periosteal osteo-sarcoma, and I have even found them in muscular sarcomata. On account of their size they sometimes give to a tissue an alveolated appearance, and by softening lead to the formation of cysts (*a*), or they may ossify (*b*).

It has been proved by the researches of Kölliker and Wegener that these giant cells occur very frequently during the absorption of bony tissue, and it has also been stated that they often form the nucleus of a smallest tubercle. They occur, therefore, not in these tumours alone, as has been imagined by some, but elsewhere in such quantities and are so highly developed, that it appears justifiable to name a variety of sarcoma after them.

(D) *Reticulated sarcoma*. *Mucoid sarcoma* (gelatinous sarcoma of Rokitansky).—If the processes of the cells are well developed, and if they should be clearly visible, there must naturally be plenty of soft transparent intercellular substance. Hence the sarcomata

FIG. 145.



Mucous tissue from a myxo-sarcoma of the scalp. Magnified 400 diameters.

with gelatinous or mucoid intercellular substance best show these stellate cells. This, however, is not always the case (Fig. 145).

There are also granulation sarcomata which may be regarded as mucoid or gelatinous tumours. If, because they present a gelatinous pulpy appearance, we should wish to group together all tumours which contain much mucus, we may call them myxomata (Virchow), or we may retain the old name collonema (J. Müller) from "colla," glue. The true mucous tissue of Virchow undoubtedly belongs to the developmental sĕries of the connective tissues (Fig. 146).

FIG. 146.

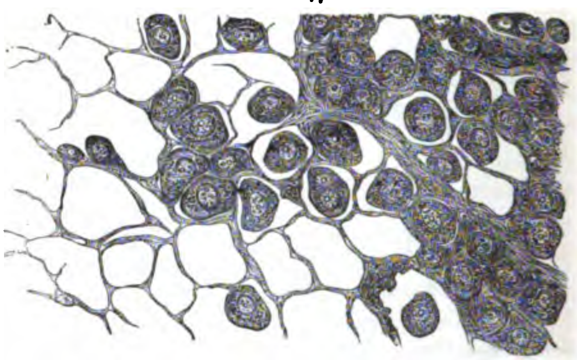
Mucous tissue from a myoma of the breast. Magnified 400 times.

It sometimes also occurs in mucoid granulations. Often enough both round and spindle cells may be found in the myxomata, and if at the same time well-developed cartilage is found the mucous tissue can be regarded as young or softened cartilage-tissue, a view which becomes the more probable if the myxoma should contain honeycomb spaces such as are found in chondromata. We may, for convenience, use the compound expressions myxo-sarcoma and myxo-chondroma.

(E) *Alveolar sarcoma.*—This, on the whole rather rare, form of tumour (it occurs in muscle, in the skin, and in bone) is difficult to characterise anatomically; it may in places so closely resemble carcinoma in the size and arrangement of its cells, that I could not undertake offhand to decide rightly the nature of any piece from such a tumour under the microscope. The elementary cells are much larger than lymph-cells; they are about as large as cartilage

cells, or moderately sized epithelial (pavement) cells; and generally have one or more large nuclei, with bright nucleoli. These cells are embedded in an intercellular substance, which is generally fibrillated, more rarely homogeneous and ill-developed, and very typically alveolar in its arrangement: the cells are generally packed singly; sometimes, however, they are arranged in groups (Figs. 147 and 148). They are intimately connected with the fibres,

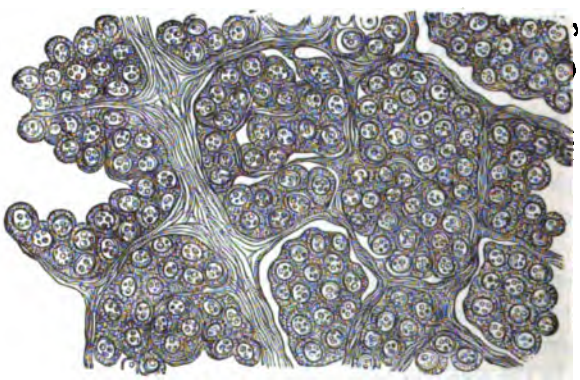
FIG. 147.



Alveolar sarcoma from the deltoid. Magnified 400 times.

and are separated from them with great difficulty. The two last peculiarities are important for the histological diagnosis of sarcoma; for they show that these large cells are connective-tissue cells and

FIG. 148.



Alveolar sarcoma from the tibia. Magnified 400 times.

not epithelial cells, as in the carcinoma. Sometimes the cellular elements of these sarcomata lie closely packed without any inter-

cellular substance: the resemblance to epithelial carcinoma may be very deceptive. Virchow has described and figured this form of sarcoma as growing from soft warts of the cutis.

(F) *Pigmented sarcoma. Melanotic sarcoma. Melanoma.*—All these names indicate that we have to do with the formation of pigment in a sarcoma. This pigment, which is mostly granular, rarely diffuse, may be either brown or black; it lies almost always in the cells; very rarely in the intercellular substance. The tumour is sometimes pigmented throughout, sometimes only in places, sometimes slightly, sometimes very strongly. Each one of the above-named varieties of sarcoma may occur pigmented, but I have seen it most frequently in the last named and in the spindle-cell varieties. Melanomata most commonly develop in the cutis, generally on the foot or hand, though sometimes also on the head, neck, and trunk.

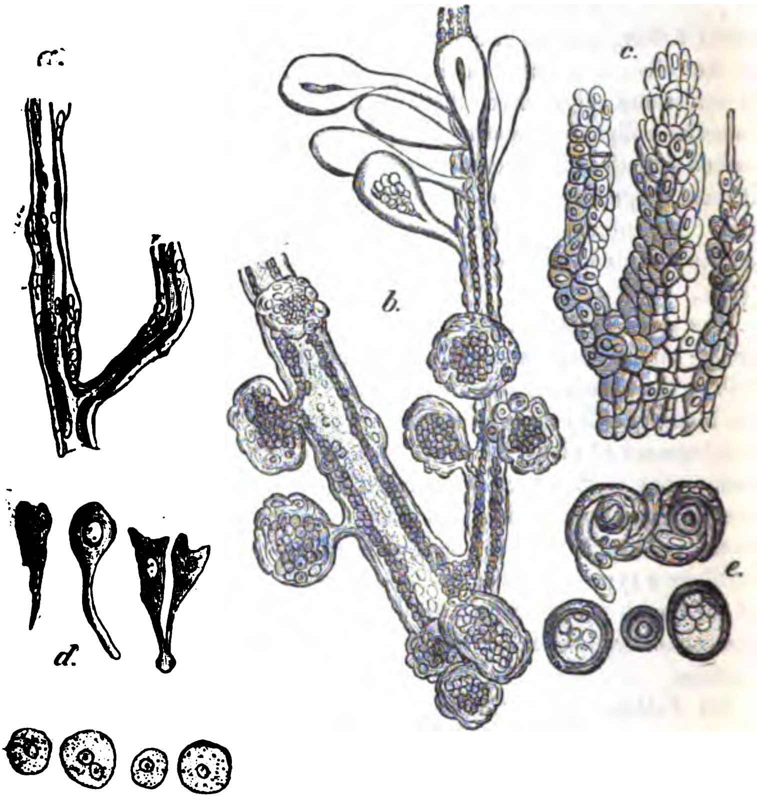
The arrangement of the cellular elements in the sarcomata depends, on the one hand, on certain directions of the fibres or fibre cells in the tumour-tissue, and, on the other, on the arrangement of the blood-vessels: in consequence of these conditions, as also of the development of giant cells or other structures, a type of tumour tissue may result which is scarcely to be distinguished from the areolar structure, which was once considered peculiar to carcinoma. This is not to be wondered at, for we have also in cartilage a type of cavities with enclosed cells, and also the network of the lymph glands, which undoubtedly belong to the system of the connective tissues, but which must be described as alveolar formations.

(G) *Villous sarcoma* (infiltrated and superficial), *pearl tumours and psammoma.*—The serous membranes, as is well known, possess the power of forming, as the result of certain pathological processes, villus-like proliferations, the stroma of which is formed of connective tissue, and ultimately of blood-vessels, while the cellular elements consist of enlarged and multiplied endothelial cells.

The extensive villi formed on the synovial membranes in arthritis deformans, the villous proliferations of the pericardium and endocardium of the valves, the granulations on the choroid plexus, and the Pacchionian glands of the membranes of the brain, are all types of this form of new growth. These particular growths, as yet, have only been found in the brain membranes and in the nerve-sheath prolongations from them; they are to be regarded as the

highest conglomerate developmental stages of the above forms; many of these growths preserve this villous appearance, at least externally, while others form more compact masses by growing dendritically into and amongst each other (see Fig. 149).

FIG. 149.



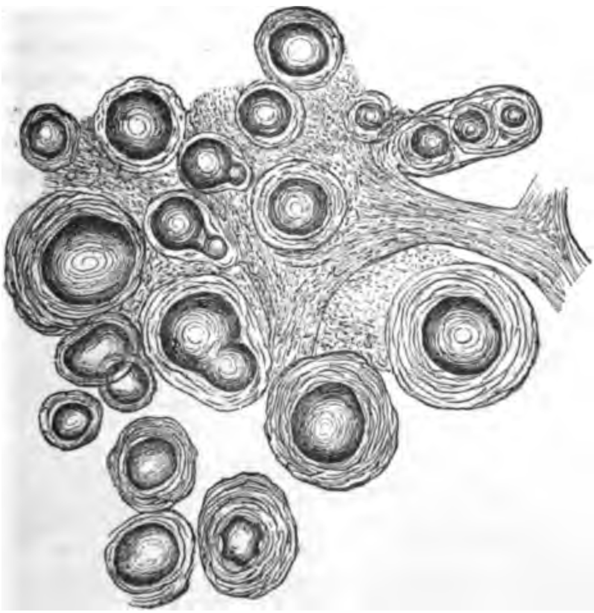
From a villous sarcoma (cancroid, according to Arndt) of the pia mater. *a.* Commencing cell-infiltration in the capillary walls. *b.* Villous bud-like proliferations growing out from the vessel walls. *c.* The same, but covered with a thick layer of endothelial cells. *d.* Endothelial cells of most perfect development, and indistinguishable from epithelial cells. *e.* Conglomeration of these cells into spherical heaps. Endothelial pearls. Multiplied 400 times.

The mode of development of these tumours is as follows:—A circumscribed cell infiltration commences in the adventitia of the blood-vessels (*a*), which leads to the knob-like villous outgrowths;

this soon becomes a hyaline or fibrillated connective tissue, or else it forms itself into a cavity, which subsequently enters into connection with the lumen of the vessels (*b*); a portion of the cells forms itself into an epithelioid form, and surrounds this knob-like outgrowth (*c*). In amongst these cell-masses are found globes which consist of flattened and compressed cells (*e*); some of them may become dried up, others, according to circumstances, may calcify.

Whether the pearl tumours (Virchow) of the membranes of the brain, which are composed of brilliant pearl-like non-vascular nodules, varying in size from a barley-corn to a pea, are formed from such endothelial globes, or whether they are true epithelial formation, I must leave undecided, as I have no personal experience of them, and as I have not lately learnt anything new concerning them.

FIG. 150.



Psammoma, after Virchow. Magnified about 200 times.

According to the earlier experiences of Virchow, these pearls form in intracranial tumours out of connective-tissue cells; thus, therefore, these tumours belong in the series of the sarcomata. The pearls from the thymus gland are the physiological type of

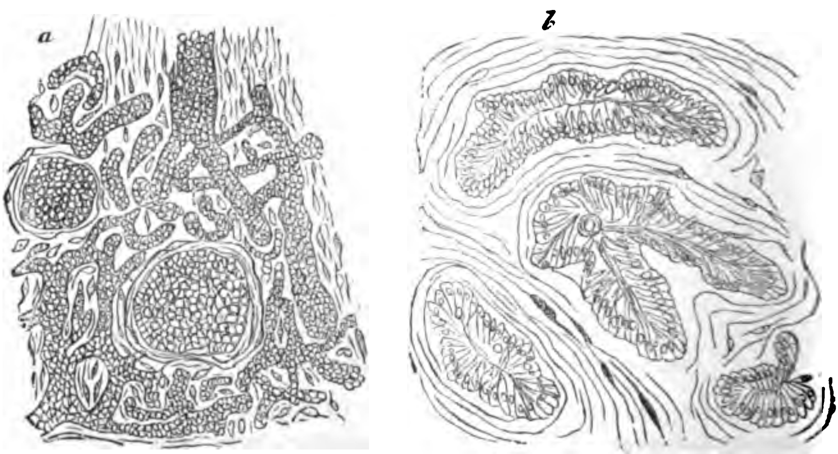
this variety, which, on account of their want of vessels, come into relation also with tubercle.

Psammoma.—A new form of tumour, first described and thus named by Virchow, belongs to this group. As yet it has only been found in the brain and orbit; it consists partly of villous and partly of the plexiform sarcoma, which will shortly be described.

This growth is characterised by the presence of calcified globes, which assume the appearance of the concretions that are normally found in the pineal glands, and known to you anatomically as brain-sand (ψαμμός, sand). Like the thymus pearls, these growths are mostly attached to vessels, and are probably in great part calcified endothelial pearls. Virchow believes that direct calcification of the connective tissue may lead to similar formations.

(H) *Plexiform sarcomata (cancroid—adenoid).*—These varieties of sarcomata are also chiefly found in the orbit and brain, sometimes in the parotid. They can only be distinguished from some of the forms of carcinoma (which we shall presently describe) by a most careful examination. Extensive plexiform cylinders, knobs, and spheres made up of small cells spread out into the surrounding connective tissue, separate the bundles from each other and fill out all the spaces between them. It is not always easy to distinguish whether the first proliferating cells are wandering connective-tissue

FIG. 151.



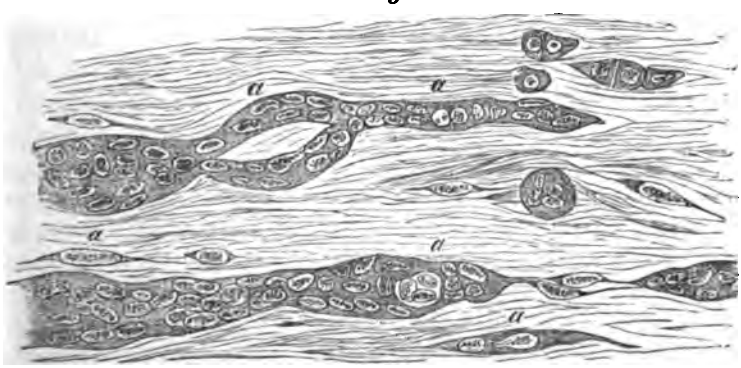
a. From a brain tumour, after Arnold. *b.* From a brain tumour, after Rindfleisch. Magnified 3—400 times.

cells or cells from the vessel walls, or endothelial or perithelial; it is possible that each of the above-mentioned elements takes part in the formation of these remarkable tissue proliferations.

The earliest cells are usually small, round, or irregularly polygonal. Gradually the following complex metamorphosis takes place in these cell cylinders:—Vessels grow into them, the central part of the cells around the vessels becomes a hyaline or fibrillated connective tissue, the external cells form a covering around the vessels and their newly formed connective filaments. Thus these formations assume, in a certain degree, a villous form, which grows into the tissue (interstitial papillary growth, interstitial papillary myxoma, Rindfleisch). While this takes place the surrounding external cells may acquire such a close resemblance to epithelial cells, both as regards shape and position, that to mistake them for sections through glands, especially under a low magnifying power, is very excusable (Fig. 151, *b*).

A very remarkable appearance is presented, if in single cylinders, the central cellular elements, in consequence of changes in their protoplasm, become fully converted into a hyaline connective tissue (Fig. 152, *a*, *a*, *a*).

FIG. 152.

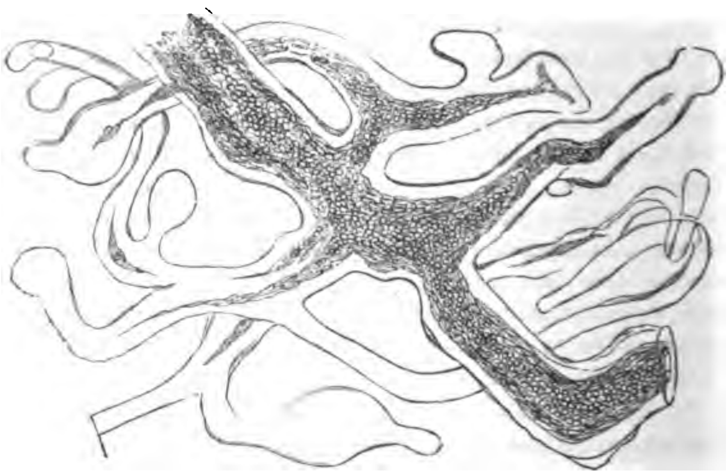


Commencing hyaline metamorphosis in the papillæ of a plexiform sarcoma. Commencement of the formation of cylinders, after Sattler. Magnified 500 times.

Then there result dendritic cactus-like growths, continuous one with another, surrounded with cells, but quite separate from them; vessels may grow into them, if the new growth does not originally proceed from vessels or grow around them.

These remarkable hyaline bulbs and cylinders were formerly mistaken for lymph-vessels. I early recognised this error, and

FIG. 153.



From a cylindroma (plexiform sarcoma, with hyaline vegetations) of the orbit. Magnified 300 times.

believed them to be outgrowing connective tissue, and hence I named them, on account of their form, "cylindroma." The origin of these growths, however, remained very obscure. I considered these cylinders, consisting, as they do, either entirely or in great part of cells, to be gland-like growths; thus the mode of development of these new growths, both to myself and some other workers who had opportunities for studying them, remained for a while very doubtful, and I long remained uncertain as to their signification and their genetic connections. It was only through the researches of Sattler that we got some real light in this matter. The above-given description I believe to be the right one, the more so because it gives the key to the interpretation of the many varieties of these growths.

If we now come to the *naked-eye appearances of* the sarcomata we must first of all notice that these tumours, in a large majority of cases, have a very sharply defined rounded outline, and that they are generally definitely encapsuled. This is an important differentially diagnostic symptom as opposed to the infiltration of carcinoma. Sarcoma only rarely affects the surface (either free or closed mem-

branous sacs) in a papillary or polypoid form, and yet there are glandless polypi both of the nose and uterus, as well as soft warts of the skin and mucous membranes, which, according to their histological structure, can only be classed in the sarcoma series. Finally, we may occasionally see infiltrating sarcomata; the villous and plexiform sarcomata not seldom appear as infiltration of the tissue. The consistence and colour of the sarcomata are so manifold and various that one cannot possibly generalise, for the greatest differences exist. There are very firm, even cartilaginous sarcomata, and others of a soft gelatinous, almost liquid consistence. The colour on section may be light rose, white, yellowish brown, grey, black, or dark red; indeed, all these colours in various shades may occur in different sections of the same tumour. This depends, except in the case of pigment, especially on the vascular supply of the tissue, and somewhat also on the amount, if any, of extravasated blood in various stages and of various dates. The amount of vascularity is extremely variable; sometimes there is only a very sparse supply, while in other cases the tumour is like a sponge and full of dilated veins (cavernous telangiectatic sarcoma). We must mention one more peculiarity of sarcomata, namely, that they are sometimes so white, and at the same time so soft, as to have the greatest resemblance to brain substance. These medullary (encephaloid) sarcomata possess the malignant qualities of sarcomata in the very highest degree, and are on that account much dreaded; they may still have any of the histological structures previously referred to. Tumours which are easily torn into bundles in certain directions, in addition to the name of sarcoma fasciculatum, have been called "bundle sarcoma" (formerly carcinoma fasciculatum.) The anatomical metamorphoses which may take place in sarcomata are manifold; the various kinds of softening are the most usual; mucoid softening with the formation of mucous cysts, fatty and caseous degenerations, are frequent. In the sarcomata occurring in connection with bone, ossification is common, and it may go on until the sarcoma has become more or less completely converted into an osteoma. Cicatricial atrophy scarcely ever occurs in sarcomata, and this is another important distinction from carcinoma. Ulcerative processes, occurring from within outwards like a crater, are rare. Sarcomata of the skin ulcerate early, without, however, leading to extensive destruction. The ulcerating surface of a hard sarcoma sometimes presents well-formed granulations.

The *diagnosis* of sarcoma on the living subject may be arrived at by attention to the following points. Sarcomata occur very often after some previous local irritation, especially after injuries. Scars also not infrequently become the seat of sarcoma. From irritated moles bland sarcomata may arise. Skin, muscles, nerves, bones, periosteum, more rarely glands—among these the breast and the parotid relatively often—are the seats of these tumours. Sarcomata occur least often in children, seldom in the second decade, most frequently in middle life, and very rarely in old people. According to my own observations, men and women are equally liable to its occurrence. If these tumours are not placed in immediate relation to nerve trunks, they are usually quite painless until they ulcerate. When sarcomata occur in the subcutaneous tissue or in the breast, they mostly present themselves as movable, encapsuled growths. Their growth is sometimes rapid, sometimes slow: their consistence so different, that this point cannot be relied on for diagnostic purposes.

Course and prognosis.—A sarcoma may occur singly, remain single, and after extirpation never return. A sarcoma may recur either singly or multiple after repeated extirpations, in the course of ten, twenty, thirty years, and metastatic growths may occur either in the liver or in the lungs; this disease may sometimes prove fatal in three months. Thence you see that the greatest benignity and the greatest malignity in the natural course of these growths may be united in this one group of tumours. I can assure you that two cases of sarcoma possessing the most uniform histological structure—generally, it is true, of different consistence—may run a totally different course. From this circumstance the strongest reproaches have been cast on pathological histology; it must, however, be acknowledged that the histological structure of a tumour does not always convey a proper idea of its clinical history, and therefore to reproach the anatomical structure of tumours is just as absurd as to blame us because, under the microscope, we cannot distinguish sections of a salivary gland, lachrymal gland, or mucous gland, from each other, although they have each a totally different signification in the organism. The expectation of everywhere finding a specific anatomical formation for each specific function must first be eradicated. And yet the data for a prognostic criticism of any given sarcoma are not wanting. We shall shortly speak of the localisation of these

tumours as a most important factor in prognosis; next to this, their consistence is of great moment; all firm sarcomata may be more favorably prognosed than soft ones: alveolar sarcomata have always a serious prognosis; while the soft granulation and spindle-cell sarcomata, which mostly occur in the form of medullary tumours, have the most unfavorable prognosis. The pigmented sarcomata are also very dangerous, although the firm are less so than the soft ones. Another important point for the prognosis is the rapidity of growth of the primary tumour; this is mostly in direct ratio with its consistence. Thus, if a sarcoma have required four to five years in order to attain the size of a hen's egg, the prognosis is not bad: whereas, if it have attained the size of a fist in from four to five weeks, then the prognosis is exceedingly bad. It may happen that a sarcoma develops so quickly as to be taken for a cold abscess: I have known a case in which a sarcoma of the abdominal walls grew so rapidly that at first the diagnosis was carbuncle. The patient (a woman) was quite beset with sarcomata, and died, within three months of the appearance of the primary growth, of metastases in the lungs. It does, however, sometimes happen that a slow-growing sarcoma may develop secondarily a rapidly growing one, but the contrary has not been observed. Sarcomata commonly develop in strong, well-nourished, and often remarkably healthy and fat subjects: I once saw a fine healthy, well-developed young woman, aged 18, the subject of medullary sarcoma of the breast; she died of secondary deposits in the lungs a few months after the operation for removal of the breast. In strong healthy men, and sometimes without any appreciable cause whatever, there come slightly pigmented, very vascular sarcomata of the skin, often commencing in the form of sanguineous blebs, and which in the course of from six months to two years spread over the entire body; then appear secondarily in internal organs, and in the course of a few years lead to a fatal result. The kind and mode of development of these consecutive sarcomata is very characteristic. The first tumour is, for instance, completely extirpated; some time elapses, and then either on, in, or near the old scar a new tumour appears; this also is completely removed, and again, at the seat of operation, or a little distance from it, another new tumour shows itself, and then others and others. The patient now begins to lose flesh, and further operations are no longer feasible. Marasmus sets in, and secondary growths, with corresponding symptoms, probably

show themselves in the liver or lungs, with exitus either as the result of suppuration from the primary tumours, or in consequence of disease of the internal organs. In rare cases, for instance in sarcoma of the skin over the thorax, or abdominal walls, or hinder part of the scalp, the course of the disease may go on through several decades.

The course of this disease differs from that of carcinoma in this way, that while in the latter the recurrence is in direct continuity with the original growth, in sarcoma, on the contrary, it is rather contiguous than continuous, provided, of course, that the original tumour was capable of complete extirpation. And this is very easy of explanation, because the limits of an infiltrated carcinoma are more difficult to define than are those of an encapsuled sarcoma; the latter, therefore, *ceteris paribus*, are the more easy to remove. If portions of the original tumour are left behind, then the recurrence of a sarcoma is naturally one of direct continuity. After the complete removal of a sarcoma many years may elapse between extirpation and the re-occurrence of local manifestations, and further, a sarcoma may for many years, and even all through life, be confined to one spot. I am acquainted with one case of a fibro-sarcoma of the hinder part of the scalp, in which from the appearance of the original growth until death through recurrent growths, there was an interval of twenty-three years; during this period the patient underwent five operations, and each time secured a longer interval of immunity. I extirpated a medullary sarcoma for an old woman (an alveolar, cancer-like form, see Fig. 147, p. 408) from the deltoid muscle; the wound was scarcely healed before a new growth appeared, like the first; the woman then remained healthy for four years, then further growths in the deltoid, for which another operation, probably incomplete, was undertaken; a further growth in the scar before it quite healed, and then exarticulation of the arm was performed. Recurrence took place on the pectoral and latissimus dorsi muscles, and death from secondary deposits and pleurisy. Three years ago I extirpated a large-celled melanotic sarcoma of the scalp in an old man, on whom Schuh had previously operated for a similar tumour six years before; as yet there has not been any recurrence. If amputation through the thigh be undertaken on account of sarcoma of the tibia, after some years there may be recurrence in the scar and consecutive deposits in the lungs. In such cases this local tendency to recurrence is only to be explained by the presence of infective germs or seedlings in the neighbourhood of a tumour,

if recurrence takes place early: but if years elapse between the different outbreaks of this disease, such an explanation no longer suffices, unless we agree to believe that these tumour-cells may remain quiescent in the tissues for years together, and then suddenly commence to sprout like a seed corn; who knows? I have one case of carcinoma, which seems to speak in favour of this view.

The mode of infection in sarcomata is very peculiar; I believe that I was one of the first to lay stress on the fact that sarcomata have the peculiar property of not affecting the lymph glands, or only very late in the course of the disease. The infection does not, as in cancer, take place through the lymphatic channels, but rather, if not entirely, through the veins. This is of great moment, and is greatly in favour of the views held by some authorities, that the vessels themselves are the most frequent source of sarcomata. The secondary deposits in the lungs are clearly demonstrable as embolic in their origin; it would appear that the walls of the vessels readily grow into the tumour mass, and that their lumina become closed with friable *débris*, which then quickly gets into the circulation. The number of secondary sarcomata is sometimes quite colossal, the whole pleura, the peritoneum, being sometimes literally bestrewn with them. The melanotic form appears, in this respect, to closely rival the medullary. Sometimes after slightly pigmented tumours very black ones, sometimes also very white ones, may occur as secondary growths. Lung sarcomata are mostly of the granulation-sarcoma variety. I have seen in the liver secondary and very

FIG. 154.

A central osteo-sarcoma of the ulna. From the Museum of the Berlin Klinikum.

beautiful spindle-cell pigmented sarcomata. Thus the forms of primary and secondary growths frequently and freely interchange.

c. Topography of sarcomata.—As the hitherto somewhat general remarks offer too few data for practice, it seems desirable that I

Fig. 155.

Section through the same specimen (Fig. 154).

should enter a little more at length into the nature of certain forms of sarcoma in special organs and special parts of the body.

Sarcomata frequently occur in the interior of the long bones (myeloid tumours, or central osteo-sarcomata), and these, for the most part, are giant-cell sarcomata. They develop with remarkable frequency in the lower jaw (Figs. 156 and 157), next frequently in the tibia, radius, ulna (see Figs. 154 and 155). Mucus cysts are frequently found in these tumours, as also formations of bone, either of a rounded or branched form. They present themselves as circumscribed nodules, springing from the medullary cavity, gradually invading the surface by destroying the interlying bone; they are covered by the periosteum however, which constantly goes on with the formation of new bone, hence these tumours generally possess an external shell of bone, although they may even grow to a very considerable size. The diseased bone then appears to be expanded into a bag, but its continuity, on account of the tumour, is not necessarily destroyed. If these sarcomata occur in the lower extremity, a very rich vascular supply is formed; a number of small traumatic aneurisms form, and a real aneurismatic friction sound may be heard within the tumour, so that often they have been mistaken for a true aneurism in the bone, and described as such. The cystic sarcomata and the compound cystomata, which are occasionally observed in the long bones, and especially in the lower jaw, are mostly developed from giant-cell sarcoma (Fig. 158); groups of which (giant cells) have undergone mucoid degeneration. Central osteo-sarcomata are generally solitary tumours, very rarely lead to general infection. In the lower or upper jaw they occur about the period of the

second dentition, very rarely at the first dentition; in the long bones I have only seen them in or about middle life. Most of the

Fig. 156.

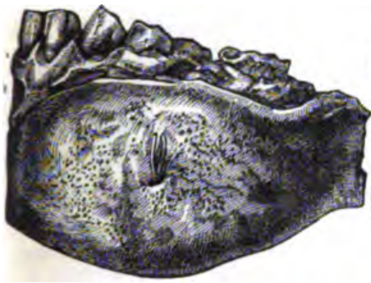


Fig. 157.

tumours which are called epulis (the word signifies "sitting on the gum" ἐπί, on, and οὖλις, gum), belong to this giant-cell variety of

Fig. 158.

Compound cystoma of the thigh, after Péan.

sarcoma. The attachment to the gum is mostly apparent; they generally come from the alveolus, and are surrounded by the granu-

lations of carious teeth roots. Epithelial cancer is by many considered as epulis. It would be better either not to use such expressions at all, or to attach qualifying adjectives to them, for instance, sarcomatous, fibromatous, carcinomatous epulis, &c. The peripheral osteosarcomata or periosteal sarcomata (osteoid chondromata, Virchow) are rather malignant; they either consist of granulation tissue with young osteoblasts, as in osteophytes, and ossify more or less completely, or of very large spindle-cell myxosarcoma with a partial ossification. The rapidity of their course is very various. Secondary deposits in the lungs have been observed.

Spindle-cell sarcoma are found especially often in muscles, fasciæ, and in the skin, which are very infectious locally, and frequently recur after extirpation. Myxosarcomata are found in the skin and subcutaneous connective tissue, and are with the naked eye difficult to distinguish from œdematous soft fibromata. Besides these, sarcomata are rather frequently found multiple in connection with nerves. The more quickly the tumours have grown, and the more medullary they appear, the more dangerous are they. I find that any period of life, except perhaps childhood, is equally disposed to these tumours.

If a sarcoma develops in a gland it almost always contains gland elements, which may be much altered in their character, and of which some may perhaps be new growth; the gland tissue seldom becomes altogether converted into sarcoma. Thus it happens that pure adenomata (which by the way are very rare) are sometimes very difficult to distinguish from sarcomata which have developed in glands.

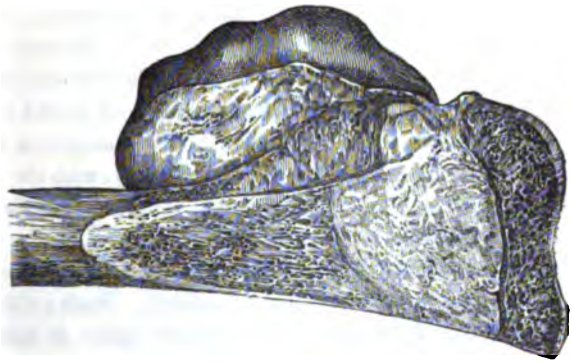
FIG. 159.

Periosteal sarcoma of the tibia from a boy. From the Surgical Museum of the Berlin Klinikum.

All glands are not equally disposed to sarcoma formation; we will briefly recapitulate the places at which it is most frequently found.

The female breast is especially prone to these tumours. Sarcomata of the breast are roundish, lobulated, and irregular tumours of an elastic consistence; the disease may attack either a larger or

Fig. 160.



Section through the same specimen.

a small portion of the gland lobules; as a rule, only one breast is attacked, and generally at one point only. In some cases, however, several small nodules may occur together in the same breast. These tumours grow very slowly indeed; they cause no pain, and, like other sarcomata, are sharply defined from the healthy surrounding tissue, and thus they are quite movable within the parenchyma of the gland. If they grow very large (and in the course of years they may grow to the size of a man's head) they almost always become cystic, and softer, and cause pain. They may ulcerate also.

The anatomical characters of these tumours have always excited a good deal of interest. As gland elements—both acini and excretory ducts—were found in these tumours, it was generally believed that they were newly formed in the tumour, and hence the growths were considered as partial hypertrophies of the breast. I consider this view to be erroneous, for I am convinced, as the result of the examination of a large number of these tumours, that it really is a primary and essential formation of sarcoma in the connective tissue between the individual acini, in which the latter are preserved, though they may undergo various changes. In consequence of distension of the glandular excretory ducts, cysts are very apt to be formed in these tumours; at first they resemble clefts, and then assume a more rounded form, and are filled with a sero-mucoid

fluid. As regards the tissue of the new growth, it is generally formed out of small round or spindle, rarely branched, cells, with a moderate amount of a fibrillated, or sometimes gelatinous intercellular substance. The fibroid quality sometimes predominates to such a degree that the whole tumour, both as regards consistence and general appearance, closely resembles a fibroma. Occasionally the development of cartilage or of bone is observed; this is, however, exceedingly rare, and exercises little or no influence on the course of the disease. If the growth of these tumours in all their parts were uniform, the excretory ducts and the acini would increase or become pressed upon in like proportion. Only imagine a portion of the gland, say one lobule, spread out superficially, and the deeper structure, to which it is firmly attached, increasing, then the epithelial surface must also increase along with it; and as the gland-ducts may be regarded as prolongations of surface with an epithelial covering, this description corresponds completely. Such an uniform growth in all parts of the tumour does not take place or only very rarely. The consequence, therefore, is that the excretory ducts alone become either lengthened or dilated; and thus the cleft-like cysts, quite obvious to the naked eye, result; while in consequence of dilatation of the glandular acini, roundish cysts are developed. As a consequence of the dilatation of these sacculated gland-surfaces, the epithelium increases and goes on to a high state of development, that is, the small roundish epithelial cells of the acini become greatly increased in number, and changed into layers of cylindrical cells. The gland substance, thus modified, pours out a muco-serous secretion, which to a very small extent is got rid of through the nipple ducts, the greater portion remaining behind in the tumour, and giving rise to the dilated spaces just referred to (retention or secretion cysts). Then a further growth of the tumour mass takes place in the form of lobulated leaflike proliferations (cysto-sarcoma phyllodes, from φύλλον, a leaf) into the interior of these cysts, so that the appearance of one of them on section is rather complicated.

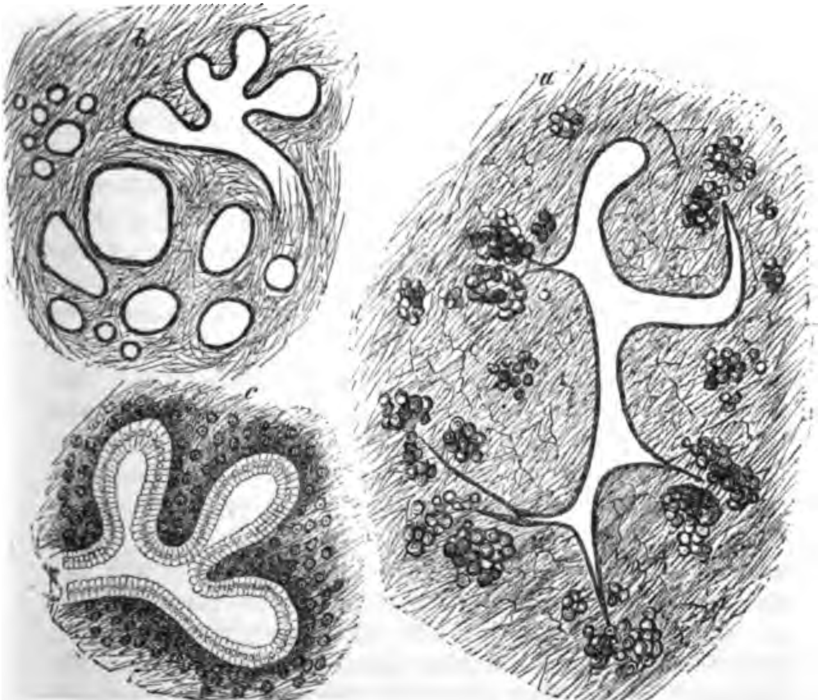
The relationship between the formation of cysts and the sarcoma tissue varies in this, as in all cysto-sarcomata very greatly, and it does not materially influence either its nature or course.

Whether plexiform sarcomata occur in the breast I am unable to say definitely, as I have had no cases since I began more particularly to study this subject.

Sarcoma of the breast and cysto-sarcoma are not very uncommon,

although in comparison with cancer of the breast (which we shall have presently to consider) they are very much less frequent. The disease occurs most frequently in young women; it occurs also shortly before puberty, and rarely after the fortieth year. The growth of these tumours is very slow, and so long as they are not very large, they are painless; later, however, there is shooting pain, and as the tumours may grow to the size of a man's head, and may ulcerate, the troubles which they may give rise to are very considerable. Some of these sarcomata possess the peculiarity that they enlarge slightly and become rather painful shortly before and during menstruation. The general health condition offers nothing very peculiar in this disease; it is only when the tumours are large and commence to ulcerate that the patients

Fig. 161.



From an adeno-sarcoma of the female breast; *a*, dilatation of excretory ducts; *b*, of the acini. Multiplied 60. *c*. A dilated breast acinus with cylindrical epithelium granulation-like interacinous tissue. Multiplied 350.

waste, become anæmic, and look ill. The course of the disease

may vary; there is a by no means small proportion of cases, in which small sarcomatous nodules in the breast, possibly originated immediately after the first confinement, and in the course of time spontaneously disappeared or that have remained harmless throughout the whole of life; but in the majority of cases, however, the tumours grow gradually until they are operated on; if this is not done until late, and until the tumour has attained a large size, and the women are old, then these growths become infectious. In young girls and young women a slow-growing sarcoma of the breast does not usually recur after operation. But if the sarcoma has not appeared until between the thirtieth and fortieth year of life, there is reason to fear general infection, and besides this, a transformation into cancer in consequence of epithelial proliferation becomes possible. I consider it advisable in all cases to extirpate the mammary glandular sarcomata early, as one cannot possibly know how these tumours, in their further course, may conduct themselves.

The diagnosis is often exceedingly difficult; small nodulated and lobulated indurations may occur in the milk-glands from a chronic inflammatory process, and especially during and after lactation, and there go away spontaneously or after the application of iodine paint. Whether, in any given case, we have to deal with a chronic inflammation which is capable of subsiding, or with a real tumour formation, can only be decided by the course which the disease has taken, and by remembering that chronic inflammatory changes in the breast are exceedingly uncommon. The most careful histological examination of the diseased structure affords next to no help; for young sarcoma tissue is indistinguishable from inflammatory new growth. Here is another instance where the boundaries between chronic inflammatory new growth and tumour cannot be accurately determined. Sarcoma often develops in the salivary glands; the tumours which form in this part are usually of a firm elastic consistence; they are movable in the gland, and have an exceedingly slow growth; they occur more frequently in the parotid than in the submaxillary gland, and are very rare in the sublingual gland. Their anatomical characters, as observed with the naked eye, are very various; the tumour is always surrounded by a definite capsule, which is in very close relation with the gland structure.

The substance of the tumour may either be almost diffluent,

cartilaginous, or fibrous, with patches of ossification or calcification; cysts, with yellowish, gelatinous, or serous fluid, are also often found in their interior.

The histological examination of these growths shows that their softer portions are made up of spindle or stellate cells, with an intercellular substance, which is present in varying degrees, sometimes fibrillated, sometimes mucoid, and sometimes cartilaginous; in addition there are the newly-formed tubular ducts. The cysts result partly from mucoid softening of the sarcomatous tissue, partly from dilatation of the newly-formed tubules. In rare cases the whole tumour is composed almost entirely of cartilage, though there is generally a small amount of sarcoma tissue. What one believes to be gland tissue in these tumours, on closer examination generally turns out to be plexiform and interstitial sarcoma formation (Fig. 151), as Sattler, who has carefully examined many of these tumours for me, has clearly demonstrated.

The tumours may develop at any time from puberty to about the fortieth year of life; they are quite painless, and grow very slowly, especially when they occur in middle life. Although they never retrogress, nevertheless small tumours of this kind, after growing to the size of a hen's egg, may in late life cease to grow. If they are extirpated in young people, as a rule they do not recur. But if removed in later years they frequently do recur, and with such rapidity too that they gradually grow into the deeper structures of the neck, and thus get beyond the reach of the knife; the nearest lymphatic glands of the neck also become affected, and then the whole course of the disease comes more and more to resemble that of carcinosis. From the above-described course of these tumours must be inferred that, as a rule, they ought to be early extirpated.

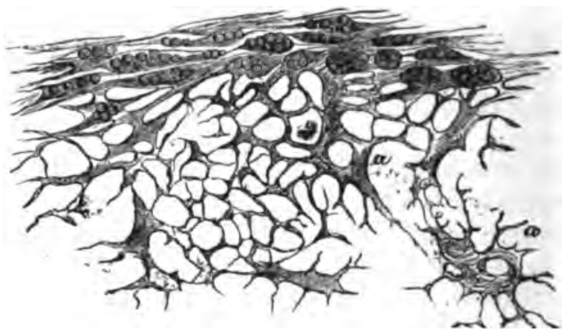
9. *Lymphoma.*

These new growths are extremely difficult to define, both pathologically and clinically. We may consider them as originating either in consequence of some inflammatory and secondary swelling of the lymphatic glands, or as an idiopathic hyperplasia. In diseases from the most varied causes the lymph glands almost always have an identical appearance; they are enlarged, more juicy, and harder than normal.

The microscopic examination of lymphoma shows the following

characters if the glands are previously hardened in some suitable fluid: All the cellular elements of the gland are increased and enlarged, the lymph-cells in the alveoli, the connective tissue, the capsules of the alveoli, and the lymph-sinuses; thus the entire structure of the gland becomes changed, for the whole organ becomes a mass of lymph-cells, although generally a fine stroma remains behind; the blood-vessels too become thickened, often very much so (Fig. 162 *a*). The cellular infiltration may become so great that a differentiation between lymphoma and glio-sarcoma (Fig. 141) is in places very difficult. Here and there large giant-cells may be developed.

FIG. 162.



From the surface of a hyperplastic cervical lymph gland. Magnified 320 times. *a a*. Sections through vessels with thickened walls. Pencilled preparation.

On examination, the glands are found to vary in size, but their intimate structure is the same. Neither the macroscopical nor the microscopical conditions allow one to state exactly whether the causes which have predisposed to the hyperplasia were idiopathic or deuteropathic; but this much one is justified in stating, that glands which have enlarged as the result of chronic inflammation more frequently contain abscesses and cheesy contents than those glands which, apparently, have enlarged from idiopathic causes. I make use of the term "idiopathic disease of the lymph-glands" perhaps from over-conscientiousness. In many of these cases we are quite unable to detect any external peripheral irritation, although there may be much to suggest that the glands are secondarily affected; it is always possible that slight and temporary inflammatory irritations have been present, which affected

the lymphatics, but which have subsided by the time the patient first comes under observation. For such residuary, secondary hyperplastic processes of the lymph-glands we have freely used the general expression of "scrofulous diathesis," and hence have called these lymphomata "scrofulous glands" (or scrofulous sarcoma, B. von Langenbeck). Let us consider them, pathologically as well as clinically, somewhat more carefully.

For some time the kidney-like shape of the glands is on the whole preserved; finally, however, this disappears as they increase in size, until at last the adjoining glands fuse into one lobulated tumour mass. Looked at with the naked eye, the extirpated glands are of a roundish, oval, or kidney form; on section they are of a light greyish-yellow colour, which on exposure to the air changes to a yellowish red. The consistence of these tumours is firmly elastic. They are easily diagnosed by their position.

The lymphatic glands are not all equally disposed to this disease; the glands of the neck are the most disposed, and this may occur either on one side or both sides; more rarely the axillary glands and the inguinal glands may become affected; the least disposed to this disease are the abdominal and the bronchial glands. The glands are scarcely ever found congenitally diseased; but disease may appear at any time from the first to the sixtieth year, although the most frequent age is between eight and twenty. This hyperplasia comes on not infrequently in several glands at once. A single gland, or several of them, in the neck may become diseased. This disposition to such new growths runs out after some years, in consequence of which tumours, which have grown painlessly and so remained, may cease to grow and may remain stationary for the rest of life. In rare cases the hyperplasia may commence simultaneously in all the glands of the neck on one side or on both, so that the neck is thickened and the movements of the head are very much interfered with. If the tumours continue to grow persistently, compression of the trachea may be caused, and death result from suffocation. Even in these severe cases a spontaneous arrest in their growth sometimes takes place, and then even large masses of the tumour can be successfully extirpated. Many of these glands may also be destroyed by chronic ulceration and caseation.

The most unfavorable cases are the medullary tumours which grow rapidly and attain the size even of a man's head (not rarely under the form of fungating masses), and in which the neigh-

bouring parts are converted into a lymphoid tissue. Patients with these tumours seldom survive; considerable anæmia comes on, the nutrition gets low, enlargement of the spleen succeeds, and death results from intense anæmia and marasmus. These malignant lymphomata, described by Lücke as lympho-sarcomata, are not distinguishable pathologically, either in their early or later stages, from the benign forms. Nevertheless they may be thus recognised, in that they grow very rapidly, and early become amalgamated with their immediate surroundings. They possess, as it appears to me, an unconquerable power of recurrence, and must be classed among the most dangerous tumours. I have recently seen a number of cases in which, at the *post-mortem* examination, metastatic lymphomata were found both in the lungs and spleen.

In some cases of extensive lyphoma very marked leucocythæmia has been observed, and Virchow believes that this increase in the number of colourless corpuscles in the blood depends on the excess of corpuscles which get into the blood from these hyperplastic lymph-glands. I do not accept this view entirely, because, after all, this leucocythæmia, even in cases of extensive glandular swellings, is on the whole uncommon, and secondly because it is highly improbable that the lymph-glands, after the complete destruction of their normal structure, can continue to perform their physiological functions. And as already a series of attempts has been made by Frey, O. Weber, and myself to inject the lymph-vessels of such lymph-glands, and as these attempts have completely or partially failed (although such negative injection-results must be carefully judged, especially in lymphatic glands), the view that these hypertrophied lymph-glands are physiologically insufficient is thus rather supported. Nevertheless, the interesting fact that leucocythæmia occurs especially in disease of the lymphatic glands and spleen is not to be denied, although its connection is not such a direct one but that some other factors, at present unknown, must be added to diseased lymphatics and spleen. Leucocythæmia has very lately been shown to stand in a nearer relation to disease of the medulla of bones, where, according to the views of these observers, the conversion of the colourless into the coloured corpuscles normally takes place; hence, then, leucocythæmia results, when from any cause, in the medulla, this change no longer goes on.

The *prognosis* of lymphoma, after what has been said, is very

various, and it is only after a period of observation as to the rapidity of growth that one can prognose with any degree of certainty. On the whole, one may assume that the disease is the more dangerous, as the patient is younger and the disease more or less extensive. I have seldom seen it originate after the thirtieth year, and I once used to believe that it never occurred after this age. Nevertheless, only a short time ago I had a patient—a fat woman, aged forty-five, who had died of asthma—in whom, at the *post-mortem* examination, a large lymphoma of the bronchial glands was found in its purest form. This tumour had ultimately caused symptoms of suffocation; and I remember another case in which an immense lymphoma of the axillary glands had developed in an old man about sixty-five years of age.

A. von Winiwarter, in a recent work, has drawn a well-defined distinction between the malignant, rapidly growing lymphomata and the primary medullary sarcomata of lymph-glands (lymphosarcoma). The former generally commence in several glands of one organ, especially the neck, remain movable for a long time, become confluent, however, at last; after a while other groups of glands become affected, and finally similar tumours occur in internal organs. Two varieties can be distinguished—a softer, on section greyish red, and a firmer, fibrous, on section whiter kind; the latter is the more rapid in its course; both forms of malignant lymphoma always terminate fatally. Lympho-sarcomata are either round-celled or spindle-celled; they occur chiefly in glands; the surrounding tissues are gradually attached to the peripheral portions of the tumour, and thus little by little they become immovable; secondary deposits in the lungs and spleen take place. I consider these differences as perfectly correct in general, and founded on careful observation; but I venture to think that combinations of the two forms not infrequently occur together.

The treatment of these lymphatic diseases will at first be an internal one. Cod-liver oil and salt-water baths (Soolsalt) are useful, and, unless the constitution of the patient contraindicates it, iodides. If the anæmia is very marked iron may be given, either alone or in combination with iodine. In some rare cases recently formed lymphatic swellings may resolve. In some cases large, quickly-growing (malignantly soft) lymphomata on the two sides of the neck, in both axillæ and in the inguinal regions, have disappeared almost entirely in about eight weeks. Unfortunately, how-

ever, the number of curable cases of this disease is very small; and exactly in those cases which have gone too far for operation, and where, therefore, internal remedies would be of such great value, do these medicines generally leave us quite at fault. Indeed, I have several times recognised the injurious effects of too energetic iodine treatment on rapidly growing tumours of this kind, especially the sudden onset of softening in the greater part of the tumours, accompanied with symptoms of violent fever. Lücke has had successful results from the parenchymatous injection of tincture of iodine into these tumours. I have seen small abscesses and slight atrophic shrinking follow this treatment, but no regular progressive wasting (phthisis) of them. A similar result has attended the action of the constant current. N. Czerny has made use of parenchymatous injections of Fowler's solution. I can also speak of the favorable results obtained by this treatment: I inject daily from one to three drops. Of external remedies iodine is the most efficacious, mercury the least so. Successful results have been obtained, especially by Baum, from compression by means of special instruments constructed for each particular case. I have noticed some improvements from it—sometimes a slight decrease in size, sometimes a partial absorption, but never any absolute cure. A cure from surgical operation can only be looked for in those cases where the disease has run its course in the glands we remove, but we are often compelled, on account of pressure on the trachea or for other reasons, to remove glands which are in an active stage of growth; in such cases we must be prepared either for a local recurrence or for disease in neighbouring glands. A careful consideration of the individual circumstances of each case must decide whether an operation is at all likely to be of service or otherwise. The effects of the operation in cases where the glands are not yet adherent, and with an unbroken capsule, are borne remarkably well. I have removed as many as twenty isolated glands from the neck of the same person—that is, I have scooped them out, like potatoes, with the fingers, without trouble and without having any recurrence. When, however, the glands have fused into a single mass of tumour-growth, and are very soft, then not only is this a sign of rapid growth and that there will be almost certain recurrence, but one may also anticipate that the operation will in consequence be very much more difficult to carry out. There are medullary lymphomata occurring in young and, for the most part, otherwise healthy men, which

spring from the deeper parts of the neck, then grow backwards behind the angle of the jaw and into the fauces, and finally affect both the tonsils and the pharynx; as a rule death takes place early. In these cases operations, though possible, are dangerous, but do not prolong life very much. Of the remaining glands, which, according to most recent researches, belong to the lymphatic system, the tonsils are the only ones which are subject to hyperplastic disease; but this hypertrophy, so common in children and young persons, belongs rather to the chronic inflammatory variety of lymphatic swelling; it results most often from chronic catarrh of the pharynx, though the contrary is usually, but wrongly, considered to be the case; that is, the hypertrophied tonsils are regarded as the cause of the pharyngeal catarrh; hence extirpation of them in such cases is of no avail, so far as concerns the primary disease—frequent attacks of sore throat. Hypertrophies of the thymus also occur, but they are exceedingly rare. Analogous disease occurs in Peyer's patches and in the spleen, but it has no special interest for the surgeon.

Lymphoma occurs also in tissues which do not belong to the lymphatics. I include under lymphoma all those soft medullary tumours in which, after careful hardening and microscopic preparation, a reticulum analogous to that in lymphatic glands can be demonstrated. In this sense I have seen lymphomata in the upper jaw, in the scapula, in the cellular tissue, in the eye, &c.—tumours which, in their structure, are with difficulty separated from granulation sarcoma, especially from Virchow's glio-sarcoma, and which, on account of their peculiar look, pass as "medullary." According to my own experience, this mixing up of the above-named varieties entails no serious prognostic error, as both sorts appear to be equally malignant and equally infectious; nevertheless, the importance of a detailed examination of these tumours is not the less obvious or to be underrated. In relation to the more definite separation of cancers and sarcomata, during the past few years we have learnt differences which are clinically both important and interesting. It would have been quite impossible ten years ago to speak as definitely concerning the group of sarcomata and lymphomata as we can at present. What we now class in the group of "lymphoma" was formerly included partly in the hyperplasia of glands, partly in the sarcomata, and partly under "medullary" tumours.

LECTURE XLVIII.

10. ***Papillomata.*** 11. ***Adenomata.*** 12. ***Cysts and Cystomata. Follicular cysts of the skin and mucous membrane; cysts of new formation; cysts of the thyroid gland; ovarian cysts; blood cysts.***

10. *Papillomata. Papillary Hypertrophy.*

We have been speaking hitherto exclusively of new formations of the muscles and nerves. We now pass to the new formations in which the true epithelia, originating in the upper and lower germinal layer of the embryo, play a more or less important part.

The epithelia form an essential part of two normal structures, namely, of the papillæ of the skin and mucous membranes (villi of the intestines) and of the glands. Both furnish the physiological prototype for certain forms of tumour, of which we will term the true hyperplastic forms of the first series *papillomata*, those of the second series *adenomata;* both are occompanied by a corresponding new formation of connective tissue and vessels.

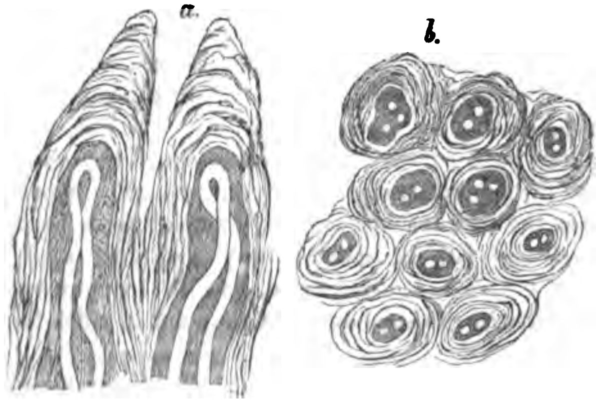
The *horny* forms of papilloma occur almost exclusively upon the cutis, seldom in the walls of the cysts of the sebaceous glands. We may distinguish two chief forms:

(*a*) *Warts.*—These are characterised anatomically by the circumstance that they arise from excessive development of the papillæ in length and thickness. Upon these abnormally large papillæ the epidermis then becomes horny in the form of small prominences, of which the wart is composed (Fig 163). These warts, which often occur extensively, especially upon the hands, without any known cause, are seldom larger than lentils or peas.

(*b*) *Horns upon the skin* are, to a certain extent, enlarged warts; the epidermic portion of the enlarged papillæ coheres to form a firm substance which increases to an enormous extent, so that

the horn, whether it be straight or twisted, may attain a length of three to four inches or more. Although the external character of these horns, which consist of horny epidermis cells only, closely

FIG. 163.



A wart. *a*. Longitudinal section. *b*. Transverse section. Magnified 20 times.

resembles that of the horns of many animals, their anatomical structure is, nevertheless, a different one, since the groundwork of the horns of animals is a bony substance. The colour of these horns upon the skin is generally dirty brown; these remarkable formations occur chiefly on the face and head, but also on the penis and on other parts of the body, and they sometimes have their starting-point in atheromatous cysts.

A general tendency of the skin is decidedly at the bottom of these warty and horny formations. This declares itself chiefly thereby that the warts appear not unfrequently in masses of twenty to fifty on both hands, especially a short time before puberty becomes developed. Irritating external influences are evidently at work here, as appears, for instance, from the circumstance that the hands are exposed to many such; that the epidermis of the hands is especially thick normally is a fact which may also predispose to these formations upon them. The disposition to horny formations upon the skin, rarely as it has been observed, belongs rather to advanced age, as do also predominantly most of the epidermoidal new formations to be described later on. In an anatomical point of view, we should add to the forms of exuberant horny growth just mentioned *hystricismus*, a porcupine-like formation of the skin,

consisting of a particular kind of papillary hypertrophy with a horny condition of the epidermis of such a kind that prickly formations become developed upon the surface of the skin. This affection, like *ichthyosis* (a scaly thickening of the epidermis over the whole body), is, for the most part, congenital.

The predisposition to warts is not at all dangerous, shows itself only in young people, and ceases quite spontaneously in many cases. It is a popular belief that warts are contagious, and perhaps not altogether an incorrect one. I saw a case in which a common wart having formed at the side of one of the toes, one also formed on the opposed surface of the next toe. The importance of horns upon the skin is much greater; although these horns sometimes break and fall off spontaneously, they grow again unless something is done to prevent them, and in many cases there even arises later on epithelial cancer at the point at which they were situated.

In the majority of cases, warts do not call for any interference. As in the case of all diseases which die out of themselves in the course of time, there are a great number of popular sympathetic remedies for warts also: the laying of such a hand covered with warts upon that of a dead person, or enveloping the hand in various leaves and plants, is regarded as an infallible remedy by old women. If you wish to remove larger warts which are particularly inconvenient and disagreeable, you may do so most easily with corrosive applications. I employ for that purpose fuming nitric acid or sulphuric acid; I touch the wart therewith, slice off the corroded layer on the following day with a knife until a drop of blood appears, and then repeat the application. This procedure must be continued until the wart has disappeared entirely.

The horns upon the skin can only be removed radically by cutting out the piece of skin upon which they are situated.

Under the name of *soft sarcomatous papillomata* we will recognise such new growths only as have the form of papillæ, consist of soft connective or sarcomatous tissue, and are covered with a layer of epithelium analogous to that of their floor.

Sarcomatous, sometimes highly vascular papillomata (soft warts) occur rarely upon the cutis, but sometimes congenitally as cock's-comb shaped exuberant growths on one or the other half of the face, almost always on one side only. Broad and also pointed

condylomata upon the mucous membranes are products of syphilis and of the specifically irritating pus of gonorrhœa; we do not count these amongst tumours in the more special clinical signification.

Sarcomatous papillomata become developed much more frequently upon the mucous membranes, especially upon the portio vaginalis, more rarely in the mucous membranes of the rectum and nose. According to the surgical nomenclature hitherto in use, they fall into the category of mucous polypi. They are frequently complex tumours in which exuberant growth and ectasis of glands, formation of sarcomatous intermediate tissue and of papilloma go on together. They are, for the most part, pedunculate tumours, but sometimes a larger surface of the mucous membrane becomes diseased simultaneously.

These papillomata seldom become infectious, but they sometimes recur after extirpation. The extensive papillomata frequently observed in the larynx in children are, perhaps, always of syphilitic origin.

11. *Adenoma. Partial glandular hypertrophy.*

New formation of true, regularly developed glands, or portions of glands, is by no means frequent, whereas we shall become acquainted later on with imperfect glandular formations in connection with cancer as one of the most usual forms of new growth.

While sarcoma of the *mammæ* was frequently spoken of as partial hyperplasia of the gland because glands were found in it, it has been much doubted more recently whether, in the cases of sarcoma in glands described formerly, glandular acini really become formed afresh; according to my observations, it would appear that true adenoma of the mammary gland occurs *very rarely*. Förster and others describe acinous adenoma of the mammæ. Their rare occurrence renders it difficult to say much concerning the prognostic importance of these tumours, which seldom attain any considerable size. They are regarded, for the most part, as altogether benignant, but it appears to me probable, on anatomical grounds, that they are not very far removed from carcinomata as regards prognosis.

The so-called hypertrophy of the *prostate*, so far as my investigations extend, is never connected with formation of adenoma, but only with ectasis of the acini and epithelial hyperplasia; the so frequently observed enlargement of these glands depends essen-

tially, as already remarked, upon diffused or nodular formation of myoma.

The glands of the *external skin* and of *several of the mucous membranes* may also give rise to the development of adenomata and adeno-sarcomata; it is believed that tumours may form upon the skin from sprouting of the glandular epithelium analogous to the glandular development in the fœtus, which are to be regarded as true adenomata. Verneuil was the first to describe a case of adenoma of the sweat glands. I have not yet seen any such tumours, but no longer doubt their existence, since it has been demonstrated by Rindfleisch. Somewhat more frequent are the glandular formations which occur in the mucous membrane of the nose, the colon, and the uterus, and which are imbedded in a gelatino-œdematous connective tissue, more rarely in other forms of sarcomatous tissue. Tumours thus form to which the name of *mucous polypi* is generally given; these are partly broad and situated immediately upon the surface, partly pedunculated; they have the colour and consistence of the mucous membrane from which they have arisen, and are covered by the same epithelium. The only exception to this is met with in the soft polypi of the external ear, which are frequently covered in a remarkable manner with ciliary epithelium. These mucous polypi do not contain glands; they are generally wanting in polypi of the ear and in the small exuberant growths of the female urethra, the so-called urethral caruncles. The last-named new growths consist exclusively of œdematous and gelatinous connective tissue, with a covering of epithelium. Most of the mucous polypi of the nasal cavity, the colon, and especially of the rectum, consist, however, in a great measure of protruding and also of newly formed glands of the mucous membrane, whose closed ends, under certain circumstances, dilate and form mucous cysts. Mucous polypi may, therefore, in an anatomical point of view, be divided according to their glandular contents, partly into true adenomata (*e.g.* mucous polypi of the rectum in children, Fig. 164), partly into sarcomata (many mucous polypi of the nose), partly into œdematous fibromata, and partly, lastly, into myxosarcomata. The predisposition to mucous polypi extends from childhood to the fiftieth year of life. In children, the disease is confined to the rectum and large intestine, and in such a manner that sometimes single tumours of that nature, sometimes many such become formed at the same time; the latter is, however, almost more frequently the

case in adults than in children. From the age of puberty until about the thirtieth year of life, the disease occurs predominantly in the mucous membrane of the nose, partly with production of single

FIG. 164.

From a mucous polypus (adenoma) in the rectum of a child. Magnified 60 times.

polypi, partly with simultaneous exuberance in both nostrils; the latter is the more frequent case. In the two last years of the third decade, mucous polypi of the uterus manifest themselves which may, under certain circumstances, furnish the transition later on to cancerous formations. All these polypi manifest an obstinate tendency to local relapses, especially in the case of polypi in the nose, the growth of which often ceases only after they have been removed three or four times. In the majority of cases, the predisposition to these new formations ceases of itself in the course of years, either because no more relapses take place, or because the smaller polypi, of the uterus, for instance, become arrested in their growth. Microscopical examination of these tumours may so far throw light upon the probable course and the prognosis that those tumours which consist of œdematous connective tissue alone are much less likely to recur than those which consist of spindle-shaped cellular tissue, or of a tissue

analogous to that of inflammatory new growth. It may happen in some cases also that an anatomical examination alone will enable us to distinguish these tumours from epithelial carcinoma.

Mucous polypi of the nose may be removed most easily by tearing them out with the forceps made for that purpose, and the same process is applicable to polypi of the external ear. Polypi of the uterus and rectum are to be cut off at their base with the scissors; if hæmorrhage is to be feared, a ligature should be applied beforehand or the écraseur employed.

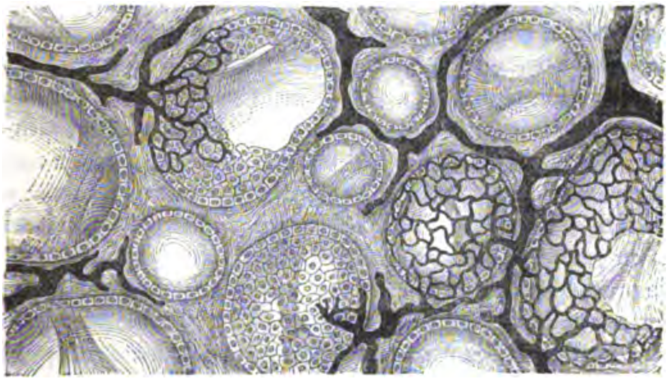
Of the glands without excretory ducts we take into consideration here the *thyroid gland* only, because it is a true epithelial gland; adenomata of the *ovary* pass so very frequently into the cystoid forms that they will be more appropriately spoken of in the next section. Tumours of the thyroid gland have long been called *struma, goître* (Kropf), the term "strumous" being used in the middle ages in the same sense as "scrofulous" at present. If we consider the anatomical relation of these tumours to the gland itself, we meet with diffused swellings of the thyroid gland which affect one lobe only, or both lobes simultaneously, and tumours distinctly circumscribed situated in the gland, while the latter continues normal or may be slightly hypertrophied. If we leave out simple cysts of the thyroid gland, the so-called struma cystica, most of the other forms of goître are true adenomata, or cysto-adenomata. Should the tissue of these tumours, which may vary extremely in consistence, not have become metamorphosed from secondary changes, it presents to the naked eye on section almost the same appearance as the cut surface of the normal thyroid gland.

Microscopically also the state of things is very similar; almost all tumours of the thyroid gland admit, when examined with the microscope, of the recognition of a great number of capsules of connective tissue, which contain a gelatinous substance studded with more or less round pale cells (Fig. 165). The size of the capsules varies greatly, since the youngest of them, which do not yet contain any gelatine, but only cells, are analogous to the fœtal thyroideal vesicles, while the diameter of the largest is from six to ten times as great.

One of the most frequent changes in tumours of the thyroid gland is the formation of cysts, which results from the circumstance that, during the dilatation of the vesicles of the gland, a number of them become confluent and their gelatinous contents more and more fluid.

Besides this formation of cysts in the bronchoceles, there are other equally frequent changes in them, which occur pretty regularly after they have existed for a considerable time, namely, extravasations of

FIG. 165.



From an ordinary, firm bronchocele. Adenoma of the thyroid gland, partially injected. Magnified 100 times.

blood, which for the most part become reabsorbed, it is true, but which leave behind them pigmented discolorations to a greater or less extent. In addition to this, cheesy and fatty degeneration, with formation of crystals of cholesterine, frequently occur in bronchoceles of long standing; lastly, calcification not unfrequently occurs, so that the original character of the tumour may become much changed by all these secondary metamorphoses. These enlargements of the thyroid gland, which take place partly in the middle of the throat, partly on both sides of it, singly or in greater number, may attain a considerable size, compress the trachea forcibly, and prove fatal by suffocation. This form of hypertrophy of the thyroid gland on both sides much more rarely attains such a high degree as to be dangerous to life. This disease is chiefly remarkable on account of its occurring endemically. There are certain mountainous countries in which it is met with: in the Harz, in Thuringia, in the mountainous parts of Silesia and Bohemia, and in the Alps, although not in all parts with equal frequency. Individual valleys of Switzerland and of the Austrain Alps are, indeed, entirely free from it. In the Himalayan mountains and in the hilly parts of the Brazils, goître occurs frequently. The most varied circumstances, especially the water and the soil, have been accused

as causes of this disease, without the possibility, however, of establishing any distinct scientific proof thereof by means of minute investigations. There can be no doubt, however, that the geological and climatic conditions play a great part in this disease. A pervading uniformity or perhaps also hereditary character in the constitution of persons affected with goître can scarcely be assumed, but a certain connection with cretinism cannot fail to be recognisable, since the majority of cretins have bronchocele also; the latter, however, is still more frequently met with in individuals with perfectly well-developed bones and brains. The bronchocele may, in very rare cases, be congenital, but generally does not become much more fully developed until the commencement of puberty; it seldom goes on growing beyond the fiftieth year. Such tumours, if they have proved harmless up to that age, usually then remain stationary and cause no further trouble later on, but there are a few exceptions to this in which a cancerous bronchocele becomes developed from the hyperplastic tumour with infection of the nearest lymphatic glands; this almost always terminates fatally by suffocation. It is scarcely necessary to speak of *struma aneurismatica* as a separate form, since it is nothing more than bronchocele with great dilatation of the arteries leading to it.

The remedies usually employed against this disease are the preparations of iodine, but these only possess real efficacy during its first development. Later on they are of no use, but are, nevertheless, employed internally and externally because we have no others. The extirpation of a hypertrophic thyroid gland, as well as that of very large bronchoceles, is always dangerous; it may cause death rapidly by hæmorrhage, or also, sometimes, in consequence of the colossal operative interference, so that it can only be thought of in the case of small, movable bronchoceles in young subjects. It requires some experience to enable us to decide beforehand whether a given bronchocele may be extirpated without danger or not. In general, I warn you against undertaking this operation for the mere purpose of improving the appearance of the patient; if there is danger of suffocation, we are of course compelled to risk more. The best prospects are afforded by movable tumours in the median line of the neck in young subjects, while even small bronchoceles embedded deeply in the hypertrophied lateral lobes cannot be removed without difficulty and danger. Even the slightest operations of this kind must be carried out with the greatest care,

especially with regard to checking the hæmorrhage from the arteries and veins (before dividing them); it is preferable, in bringing forward the encysted tumour, to use the hands, a hollow probe, or some other blunt instrument rather than to make too much use of the knife or scissors. In consequence of a judicious selection of cases, the results of my operations for bronchocele have gone on improving steadily. Kocher also has reported a series of operations of this kind, terminating favorably. Lücke, Störk, and Schwalbe speak very highly of parenchymatous injections of tincture of iodine and also of pure alcohol into these tumours; a considerable, sometimes total, shrinking up of the struma is said to follow. In the first cases in which I employed this method of parenchymatous injection of tincture of iodine it had no effect; a case in which I injected alcohol terminated fatally with ichorous suppuration of the bronchocele and septicæmia. Quite recently I have effected considerable diminution in the size of bronchoceles in some cases by constantly repeated injections of iodine; I injected twice a week a week a syringeful (about fifteen drops) of pure tincture of iodine. This must be continued several months in succession. Some of the patients lost flesh to a considerable extent, so that this mode of treatment could scarcely be recommended for weakly and especially for tubercular subjects. I have never employed the injection of alcohol since the unfortunate case mentioned above. Störk has also informed me that the injection of alcohol sometimes produce, violent inflammatory reaction, while after injection of iodine only swelling and pain of short duration occur. It is prudent to inject at first only one third, then one half, of a syringeful, to test the individual inflammatory susceptibility.

12. *Cysts and Cystomata. Encysted Tumours.*

A tumour which consists of a sac filled with a fluid or pulp is called a cyst, or an encysted tumour. These tumours may develop themselves from sacs already formed (cysts), or they may be altogether newly formed (cystomata). If a tumour is formed of a conglomeration of many such encysted tumours, they are called "*compound cysts or cystomata.*" If cysts exist at the same time in one of the tumours just spoken of, or in carcinomata, and form an essential part of the tumour, names are formed for them such as *cysto-sarcoma*, *cysto-fibroma*, *cysto-chondroma*, *cysto-carcinoma*, &c.

Virchow classes also, as stated formerly, encysted extravasations of blood, the hæmatomata (*extravasation-cysts*) already described, further, dropsical effusions and hypersecretions of serous sacs (hydrocele, meningocele, hydrops articulorum, ganglia, &c.), as *exudation-cysts*, amongst the tumours. According to Virchow's arrangement, *retention-cysts* thus form the third category of encysted tumours. Of these, let us leave the retention-cysts of the larger canals and bladders, such as hydrops vesicæ, felleæ, processus vermiformis, tubarum uteri, to internal medicine and gynæcology, and confine ourselves to that group of tumours which Virchow places together under the name of *follicular cysts*. The glands of the external skin, as well as those of the mucous membranes, are predisposed to the formation of cysts. Cysts of the thyroid gland occupy a somewhat doubtful position between the exudation-cysts, the follicular cysts, and the cysts of new formation. The closed follicles of the lymphatic glands appear never to give rise to the formation of cysts.

Of the *glands of the cutis* it is the sebaceous glands alone from which cysts become developed; I am not aware that any description has been given of cysts of the sweat glands. The causes why the secretion collects in the sebaceous glands are (*a*) inspissation, (*b*) closure of the excretory ducts. If, from one of these causes, the secretion is retained and accumulates in the gland, the secreting surface with its acini becomes expanded into a simple globular form; the accumulated secretion furnishes a mechanical irritation for the surrounding connective tissue, which consequently becomes thickened and surrounds the secretion as a cyst. If it is possible by means of powerful pressure to empty the sac before it has attained a great size, it is usual to call such a small open cyst a "*comedo.*" If, in consequence of some irritative inflammatory process, the opening of a sebaceous gland has been closed, atrophy of the gland may indeed ensue, as probably often happens, for instance, after burns with very superficial destruction of the cutis, but in other cases, the gland continues to secrete and expands into a sac which very gradually increases in size. Such cysts filled with a fatty pulp and epidermis have received the name of "*atheroma.*" This pulp is found, when examined under the microscope, to consist of fat pellicles, fat crystals, cholesterine, and epidermis cells. It differs greatly in colour and consistence; most of the cases of atheroma occurring upon the hairy scalp at an advanced age have

as contents a dirty, grayish-brown, stinking, pulpy mass. The walls of these cysts are generally thin and consist of connective tissue; their inner surface presents, for the most part, a distinctly circumscribed rete Malpighii, and is raised in a wavy or papillary form. The contents of these cysts frequently undergo chalky metamorphosis. An atheromatous tumour may be laid open by a wound, but seldom bursts spontaneously; the pulp is ejected, the edges of the opening curl back, and the internal surface of the cyst becomes converted into a very ill-looking ulcerating surface. Except upon the head and face, where they occur frequently, these tumours are rare.

A second form of these cysts are the *dermoid cysts*, the contents of which are of a pure white colour, and consist chiefly of cholesterine with some epidermis cells (*cholesteatoma*). In the walls of these cysts are found hairs with hair-follicles, and sometimes sweat glands; the cyst is, therefore, dermoid, or very similar to skin. These cysts, which occur especially often upon the head in the neighbourhood of the orbits, are at their first commencement always congenital. They are regarded as portions of cutaneous glands which have penetrated too deeply, and become isolated and developed independently in the manner described above.

In *mucous membranes* also, thickening of the mucus of the glands, and consequent difficult discharge thereof, may give rise to the formation of mucous cysts, but closure of the excretory duct is here, probably, more frequently the cause of the development of retention-cysts. The secretion in them is generally a tough, often very thick mucus of a honey-yellow (meliceris) or reddish-yellow, or even chocolate-brown colour. On examining the contents of such a cyst with the microscope, we find numerous large, pale, chiefly round cells in homogeneous mucus, which often contain granules of fat, also crystals of cholesterine frequently in great quantity. In the mucous membrane of the nose these cysts are very rare, but are met with in mucous polypi of the nose, and indeed often in such numbers that these tumours have received the name of "cystic polypi" (Blasenpolypen). Luschka has frequently found numerous small cysts in the antrum of Highmore. In the mucous membrane of the mouth, mucous cysts occur predominantly on the inside of the lips, more rarely of the cheeks. Mucous cysts are common in the mucous membrane of the uterus, and in mucous polypi of that organ. In the mucous membrane of the rectum, on the contrary,

no mucous cysts are found, and they are extremely rare also in the mucous membranes situated more deeply in the interior of the body.

Cysts of new formation.—These arise, for the most part, from a process of softening following upon cellular infiltration of diseased tissues, or from softening of portions of firm tumours. As soon as the new formation has become divided into sac and fluid contents, a secretion begins to take place, in many cases, from the inner wall of the sac, so that the cyst resulting from softening becomes a secretion- or exudation-cyst, and enlarges as such. Every tissue rich in cells may become converted into a cyst, quite independently of any formation of mucous glands, by mucous metamorphosis of the protoplasm, or, according to the views of others, from separation of the mucous substance by cells. We are acquainted in the fœtus with a formation of cavities by mucous softening of cartilaginous tissue, namely, the development of the cavities of the joints. It is precisely in cartilaginous tumours that a mucous softening of individual parts frequently occurs and leads to the formation of chondromata with mucous cysts. In like manner, softening with formation of cysts is by no means uncommon in myxomata, and the same thing happens in sarcomata, especially in those consisting of giant cells. The cysts with very smooth walls and serous, or sero-mucous contents which are met with in myomata of the uterus, are, perhaps, enormously dilated lymphatic cavities. The cysts in bones always arise at first through softening, but it may happen that the frequently very shining smooth membranes which line such cavities may, in the course of time, really become capable of secreting.

While all the kinds of newly formed cysts just mentioned bear no relation to glandular new formations, those now to be mentioned have their starting-point in adenomata. The cysts of the thyroid gland already described (encysted bronchocele) occupy a somewhat uncertain position in this series; uncertain in so far as they do not spring directly from newly formed acini or canaliculi of the gland, but from an accumulation of a for the most part mucous secretion in one or more thyroideal vesicles; if we regard the contents of these vesicles as a secretion, as there are many grounds for doing, these cysts ought to be classed with the retention-cysts; but since it might be said, on the other hand, that it is a dubious matter to speak of secretion by the thyroid gland, since the contents of the thyroideal vesicles are spoken of by many anatomists as consisting

normally of cells only, cysts resulting from the mucous softening of the contents of the thyroideal vesicles may also be regarded as newly formed. Whichever view we take, one thing is certain, namely, that cysts of the thyroid gland may occur quite singly and attain a a great size. Moreover, there are found in every large and in many small, otherwise firm bronchoceles one or more cysts whose walls are generally very smooth. It is precisely the large and isolated cysts of this kind which convey the idea that they are predominantly secretion-cysts, while all such cavities in the remaining parts of very large bronchoceles give much more the impression, by their softened and tattered walls, of cysts resulting from softening. The process of softening in the thyroid gland generally ends in the formation of a mucous fluid, but cysts are also met with in that gland which contain a gray crumbling pulp which resembles in appearance the contents of the sebaceous glands, but differs essentially therefrom, since it consists of detritus of the tissue of the thyroid gland only; I have never seen true atheromatous pulp in the thyroid gland.

To the *complex* encysted tumours belong the *cysto-sarcomatous tumours of the mammary gland,* of which I have spoken already, *cystomata* of the *ovary* and *testicle, cysto-adenomata, cysto-sarcomata,* and *cysto-carcinomata.* According to recent investigations, it is a question, in a very large majority of cases, of newly formed glandular acini or canaliculi from which terminal knobs become detached by constriction, as occurs normally in the formation of the follicles of the thyroid gland and ovary. In these newly formed follicles (perhaps also in the normal follicles of the ovary) is secreted a mucous, yellow, brownish-red, or dark brown fluid, by which the follicle, visible at first under the microscope only, gradually becomes more and more distended. Either from such a follicle, or from the confluence of several to form a larger common cavity, colossal cysts frequently form in the ovary, which may cause greater distension of a woman's body than occurs in the ninth month of pregnancy. In other cases, hundreds or even thousands of such follicles become developed and thence arise the multilocular encysted tumours of the ovary. The latter process takes place in the testicle also, although much more rarely than in the ovary. In both these organs, as in the mammary and thyroid glands, the contents of the cysts are generally to be regarded as mucous, but in the newly formed follicular cysts of the ovary and the testicle, secretion of fat and copious

production of epidermis occasionally occur. This process then either stops with the formation of epidermis-beads of the size of millet seeds or peas, grouped together cauliflower fashion, as I have seen in tumours of the testicles, or large cysts containing a fatty pulp are formed. The walls of these cysts, which may attain the size of a child's head and are met with even larger, in rare cases, in the ovaries of elderly women, are generally much more highly organised than those of the dermoid cysts of the skin, for large numbers of hairs, sebaceous and sweat glands, papillæ, and even warty growths are found in the walls of these cysts. Even cartilaginous and bony plates, with teeth of the most varied forms, are found in them, so that the idea readily presents itself of imperfect impregnation and ovarian pregnancy.

In addition to the points already named, congenital compound encysted tumours are met with in the neighbourhood of the os sacrum also, which often contain ciliary epithelium and, besides many other tissues, occasionally also glandular and follicular formations. The manifold nature of the forms of tissue in these congenital "tumores coccygei," from the comparatively simple forms of cysto-sarcoma to the fœtus in fœtu, is enormously varied, and cannot be discussed here without entering too much into details and speculation. Virchow calls such tumours, in which entire organs or fully developed parts of organs exist "*teratomata*" (from τερας, a prodigy, or monster).

I must mention, lastly, the cysts described here and there in medical literature *which contain perfectly fluid venous blood* and have smooth walls. Many of these, when punctured, rapidly fill again, others slowly; such cysts are found in the axilla, upon the chest and upon the neck. If we exclude those cases in which effusions of blood give the colour of dark blood to the mucous or serous contents of cysts, and only take into consideration those cases in which it is really a question of blood alone as contents of the cyst, these blood-cysts can scarcely be anything else than large sacs in veins, or cavernous venous tumours. All the cases hitherto made known were cured by puncture and injection of iodine, so that nothing can be said concerning the pathological anatomy of these tumours.

The *diagnosis* of a cyst is easy if the tumour fully admits of palpation; we can feel the fluctuation; deeply seated cysts are often very difficult to recognise as such. We may confound them,

possibly, with other encysted cavities with fluid contents. A test-puncture with a very fine trocar is allowable to render the diagnosis clear when this is necessary to enable us to decide upon the treatment to be adopted. There are several things with which a cyst may be confounded, *e. g.* torpid abscesses are painless fluctuating tumours, which sometimes increase in size very slowly. Echinococci also, of which two kinds occur in external parts of the body, namely, in the subcutaneous cellular tissue, *cysticercus cellulosa* and *echinococcus hominis*, become developed, although very rarely, in that tissue (still more rarely in bone); the former is a small, the latter a larger bladder, which may contain several smaller ones. The bladder of which the creature consists is surrounded by a newly formed sac of connective tissue; the whole naturally gives the idea of an encysted tumour. I have seen bladders of cysticercus extirpated from the nose and tongue, of echinococcus from the subcutaneous cellular tissue of the back and thigh; in all these cases, the diagnosis was of cysts, in one of the latter only of abscess, and here in fact suppuration had set in around the dead echinococcus bladder. I have introduced this here merely in a supplementary way, because we are not called upon otherwise to occupy ourselves much with parasites. The trichinæ which sometimes exist in myriads in the muscles cannot become the object of surgical treatment, although the diagnosis has been rendered possible in many cases, and has even frequently been formed, thanks to the brilliant investigations of Zenker.

Dropsies of the subcutaneous bursæ and of the sheaths of the tendons, as well as spina bifida, may also easily be mistaken for encysted tumours if we do not bear in mind the anatomical position of these swellings. Cystomata may also be confounded with other soft gelatinous forms of sarcoma and carcinoma and with very soft fatty tumours. As already remarked, if a certain diagnosis is required on account of operative interference, a test-puncture may be made. What guides us chiefly, however, in our diagnosis of tumours in general is our experience concerning their comparatively more frequent occurrence in one part of the body or another. I have always given you the sum of these experiences with each form of cyst and shall also direct your special attention to this point later on in the clinique.

Since the *prognosis* of encysted tumours, which all grow slowly if they form alone as cysts without complication, is included in what

has already been said, we may pass on at once to their *treatment*. We may get rid of cysts in two ways, namely, by withdrawal of the contents and the local use of remedies which excite an inflammation leading to the shrinking up of the sac, or by the extirpation of the cyst. The latter is always the simpler operation and that by which we may most quickly effect our object, and we shall always give it the preference when it can be performed easily and without danger to life. In the case of cysts of the ovary, the thyroid gland, and others, however, which are in a deep or otherwise dangerous situation, another more harmless mode of proceeding is naturally very welcome if it offers us a fair prospect of success. We may effect a shrivelling up of the sac, after previous emptying of its contents, partly by a suppuration, partly by a milder, drier process of inflammation. If you lay open the walls of the cyst in its entire length and keep the edges of the incision apart, suppuration and formation of granulations will take place upon the exposed inner wall of the cyst, with throwing off of the elements of the tumour adhering to it, or of the epithelium; the sac then gradually shrinks cicatricially and thus first becomes diminished in size and then heals, but this process may sometimes require many months for its completion. You may also attain the same end in a more subcutaneous manner if you pass through the tumour at one or more points ligatures or tubes; in consequence of the entering air, or of the irritation caused by the ligatures or tubes passed through the cyst, suppuration and granulation may also be established in its inner wall and lead, under very favorable circumstances, to the shrinking up of the cyst. This very frequently does not follow as desired, or requires months or years, at least, for its completion, so that, of these two methods, the first is to be preferred; it is especially applicable to cysts about the neck. A shrinking up of the cysts and drying up of their contents may also be effected in a different way, namely, by puncture followed by the injection of tincture of iodine; we have already spoken of the effect of this treatment. The result of the injection of iodine consists here also therein that immediately after the injection a violent inflammation of the sac sets in, with sero-fibrinous exudation; this serum then becomes absorbed and the sac contracts. The injection of iodine is suited especially to cases in which we have not to deal with softened tissues as contents of the sac, but with a fluid secreted chiefly by the sac itself, especially, therefore, in the case of cysts containing serum and of some kinds of mucous cysts. For cysts in

the thyroid gland, we may often employ puncture and injection of iodine with brilliant success, but much depends upon the method which you adopt, of which more in the clinique. Cystomata resulting from softened gelatinous tissue and fatty cysts are not well suited for the injection of iodine, since it is frequently followed by very violent inflammation and ichorous suppuration with formation of gases, so that it may afterwards become necessary to lay open the whole sac. Sacs with very thick walls also, which cannot contract at all or only very slowly, are not suited for this treatment. This we find amongst cysts of the neck especially, many which are suited for this treatment, others which are not so on account of the too great thickness of their walls. Of the cystomata of the ovary also, but few, unfortunately, are suitable for the treatment by injection, so that quite recently the treatment of these tumours by laparotomy has come to be regarded as the only safe operative procedure, by means of which, during the last few years, more and more favorable results have been attained.

I must mention, lastly, that there are cases in which it is best to abstain from any operative interference; I should look at it as a piece of folly, for instance, to persuade an old man who has a number of atheromata upon his head, to consent to the extirpation of those tumours; the supervention of erysipelas of the head might prove fatal under such circumstances.

LECTURE XLIX.

13. *Carcinomata; history. General remarks upon their anatomical structure. Metamorphoses. Different forms. Topography:* 1. *External skin and mucous membranes with pavement epithelium.* 2. *Mammary glands.* 3. *Mucous glands with columnar epithelium.* 4. *Salivary glands aud prostate.* 5. *Thyroid gland and ovary. Treatment. Short remarks concerning the diagnosis of tumours.*

13. *Carcinomata. Cancerous tumours.*

To give you an idea how the diagnosis of tumours was formerly made, and how many of the names still in use arose, I will read you a passage relating to this point from the classical work of Lorenz Heister, who was one of the highest authorities of that day, a book which reached its third edition in 1731, and now lies before me. It is there stated (p. 220): "The name of *scirrhus* is given to a painless tumour which may form in any part of the body, but does so especially in glands, and has for its cause an obstruction or desiccation of the blood in the indurated part. P. 306: "If a *scirrhus* can neither be dispersed nor kept at a standstill, nor has been removed in good time, it becomes, either of itself or from bad treatment, malignant, *i. e.* painful and inflamed, in which state we begin to call it *cancer* or *carcinoma;* the neighbouring vessels then often swell and spread out like the feet of a crab (which does not occur, however, in all cases), whence the disease has received its name; which is in truth one of the most troublesome, cruel, and painful diseases. When the cancer is still entirely covered by the skin, it is said to be *hidden* (cancer occultus); but when the skin is broken or ulcerated, it is then called an *open* or *ulcerated cancer,* and the latter generally follows the former."

It is by no means long since the simple belief existed that such comparisons and descriptions contained something real and prac-

tically useful. Will people laugh, a hundred years hence, at our present anatomical and clinical definitions, as we do now when we read what good old Heister wrote? Who knows? Time advances with giant steps, and before we think it possible, things come to light which render the patient labours of active young investigators historical in a very short time.

We are always extremely unwilling, in natural science, to give short definitions, because this is frequently almost impossible on account of the transition from one process to another and from one formation to another. Concerning carcinomata we may say, in a clinical sense, that they are tumours which are in a high degree infectious and that this infection, which extends first of all to the lymphatic glands and eventually to other more distant organs also, is probably brought about chiefly by the carriage of certain elements (whether of cells only or of fluid is doubtful) from the tumour, by means of the lymphatic vessels and veins, into the blood.

Attempts have been made to test the correctness of this already long-established idea of carcinoma by means of the anatomical structure of these tumours, and means were sought for of distinguishing carcinomata with certainty from other tumours of similar appearance. The classical monographs of an Astley Cooper on the diseases of the testicles and mammary glands (the latter unfortunately not completed) show that by a careful study of the phenomena recognisable with the naked eye much may be attained if we confine ourselves to a given organ; a generalisation is not feasible, however, by means of anatomical preparations alone and is difficult, as I have often felt in the course of these lectures, even with our present auxiliary means. I do not find fault with Virchow, therefore, if he seeks, in his great work upon tumours, to give the minutest details possible concerning the individual forms of tumour in given localities, and willingly steers clear of the difficult general clinical questions. Here, where we must be brief for the purpose of furnishing a provisional anatomical basis for our views, we are compelled to speak somewhat more decidedly and summarily. When the naked eye no longer sufficed for the diagnosis of tumours, we called in the aid of the microscope and sought for characteristic constituents belonging in a similar manner to all those tumours, the clinical characters of which we have mentioned above. But if we looked for the characteristics of cell-elements in the size of the granules, &c., their clinical and anatomical properties would not

always be in accordance. When *cancer-cells* had proved useless as a warrant for carcinomata, the characteristic properties were sought for in the whole structure of tumours; the *alveolar structure* was to be the anatomical landmark. With this also we get into difficulties here and there: the reticular formation of newly formed lymphatic-gland tissue may also be designated as "alveolar;" but even if we admit that the lymphoma-nets are so distinctly characterised by their form as to be excluded easily, many forms of chondroma and sarcoma, especially the giant-celled and other large-celled sarcomata, forms which we have distinctly pointed out as alveolar sarcomata, and in particular the interstitial villous and plexiform sarcomata, still remain as doubles of the carcinomatous tumours. Nevertheless, even professed pathological anatomists insist upon regarding the alveolar and adenoid structure and the infiltration of the new formation as alone distinctive for the designation "carcinoma."

Since the anatomical studies, and especially the *genesis* of newly formed tissues, have been introduced by Virchow and accepted as an essential principle for classification, we are released from all the difficulties just mentioned. *The anatomical development alone now serves to determine what is to be called a cancer.* The clinical inquirer seeks to ascertain how the cancers formed and composed in a given manner behave; when they are infectious; when not; whether they run their course quickly or slowly; whether they are generally multiple or solitary; where they most frequently form, and what experience shows to be the best mode of treating them, &c., and it is very interesting to observe how, with the more and more detailed anatomical arrangement, corresponding clinical differences also become more distinct. Most of the modern pathological anatomists and surgeons agree *in calling only those tumours true carcinomata which resemble in construction the true epithelial glands* (not the lymphatic glands) *and the cells of which are derived from true epithelia.*

I am convinced that this view will gain more and more adherents, and that the differences of opinion concerning the limits of the anatomical conception "carcinoma" will cease entirely. Those investigators who, in the course of the last few years, have worked impartially; with all the modern auxiliary means, in this field of the doctrine of tumours, recognise the great importance of exuberant growths of epithelium for such tumours as we are accustomed to

call cancer, but most of them still seek to make a compromise between the various histogenetic views, and are still disposed to admit in a modified form the formation of true glandular and epithelial cells from connective tissue (heterology [Rindfleisch, Volkmann, Lücke, Eberth, Biesiadecki, Gussenbauer]), and only Thiersch, and quite recently Waldeyer, believe firmly, with me, in the strict boundary between epithelial cells and cells of connective tissue. Waldeyer, who has laboured with great success in this field, has described carcinoma as an *atypical epithelial new formation.* I must at once remark here, however, that in cancerous tumours, in addition to the epithelia, there are found also a great number of fresh, small, round cells which, being infiltrated into the portion of the tumour composed of connective tissue, form a not inconsiderable part of these new formations. This small-celled infiltration of connective tissue, which is met with in larger or smaller quantity, but everywhere where exuberant epithelial growths push themselves into the tissues, appears to result from a kind of reaction, and to be a consequence of the advance of the epithelial formations into the tissue. According to the number of the cells infiltrated and to their after fate, as well as to the degree of vascularisation, it leads, as in the process of inflammation, sometimes to softening, sometimes to shrinking and cicatricial thickening of the tissues. In many cases this small-celled infiltration becomes so considerable as to cover almost entirely the epithelial new formations, from which, if the latter are small, they cannot be distinguished without great difficulty. It may then be doubtful whether they should not be regarded as completely independent and occasionally, perhaps, the only clearly recognisable component parts of cancerous tumours. I formerly felt compelled to admit this, and also did not look upon an independent capability of infection as improbable in this component part of the carcinomata; further investigations with more modern auxiliary appliances have rendered it more and more probable to me, however, that in the smallest cancerous nodules also the epithelial elements are in proliferous action. The epithelial cells and the floor from which they grow and draw their nutriment are in very close relation to each other. There are many observations which show distinctly that the cellular infiltration of the floor of the connective tissue is followed by an increased exuberance of the epithelium covering it, and it is thus conceivable that the first impulse towards atypical adenoid exuberant growth may be fur-

nished by a state of irritation of the epithelial floor. It is, however, equally possible and probable that the exuberant epithelial growth is the first formative process in the development of carcinoma, as we are at present accustomed to assume. Direct observations on this point are impossible; the infiltration of the connective tissue always exists simultaneously with the exuberant epithelial growth, and this renders the investigation of the first stages so difficult that it is only by selecting favorable objects, *e.g.* flat cancer of the skin, that we can attain a clear conviction of the correctness of our views. Since this question was first seriously discussed, it has already recurred repeatedly, not only in individual generations but in individual minds. I cannot repeat here all that which I said formerly concerning the formation and increase of the true epithelia; all that I will add now is, that the carcinomatous and epithelial forms which are met with in primary carcinomata have also always been found in the infection-tumours of the lymphatic glands. This appears to me to speak strongly in favour of the transportation of cell-elements, for it is scarcely conceivable that fluid from a columnar epithelial cancer should be capable of causing the cells of the lymphatic glands to produce columnar epithelia.

It is especially important and difficult, in an anatomical point of view, to draw a line between adenomata and carcinomata or between the complex forms of sarcomata and carcinomata, since the various forms of tumour have much in common, and may even, abstractedly from their genesis, be perfectly similar in appearance. True adenomata are composed of newly formed glandular substance, which is completely analogous to the normal substance, or, at least, very nearly so; the connective tissue surrounding the newly formed acini behaves towards these as towards the normal, it is entirely unchanged, or to a very slight extent only infiltrated with small cells. In sarcomata in glands, no new formation of glandular acini takes place ordinarily, but the sarcomatous mass merely surrounds the glandular spaces, which may either have remained normal or become dilated, with epithelial cells which have perhaps become considerably increased in number and enlarged. But carcinoma is characterised by the circumstance that the epithelial covering of a skin or mucous membrane, or the epithelial lining of glandular cavities, grows into the tissue of the skin in an acinous or tubular form, just as occurs in the fœtus. In most cases the epithelial cells retain their form, but frequently become much larger than normal. The form of the

glands from which these growths proceed also generally remains typical for the new formation, but glandular cavities capable of secretion are seldom developed therein. In the neighbourhood of this epithelial portion of these tumours, the bones, the connective tissue, the muscles, &c., into which the epithelium has penetrated behave as follows:—the connective tissue is found to be sometimes of normal, sometimes of abnormal density, sometimes extremely soft and almost mucous, and generally in somewhat less than normal quantity. It is generally studded with small round (migrating) cells, often to such an extent that scarcely any fibrous tissue remains. When it penetrates into bone, the latter becomes consumed as in caries. I have not been able to convince myself that in the nodular and infiltrated forms of true cancer a new formation of fibres of connective tissue takes place, any more than I have found new formations of bone therein; there can be no doubt, however, that in the villous and papillary forms, to be mentioned specially later on, such a new formation does take place. You see from this description, gentlemen, that Waldeyer's assertion that the *epithelial new formation* in *carcinomata* is *atypical* (tissu hétéroadénique, Robin) is also very suitable for distinguishing carcinomata from *adenomata* as *typical* epithelial new formations. As regards the vessels in the development of carcinomata, we can convince ourselves, by artificial injection, that dilatation of the vessels and new formation by means of loops occur to a very considerable extent; *only the connective tissue portions of the tumours are vascularised, the epithelial portions remain free:* this is a very important anatomical criterion, as is also the circumstance *that the true epithelial cancer-cells never coalesce,* as appears to be the case with the large epitheloid cells of many of the sarcomata. To the latter circumstance Waldeyer has justly attached great importance. By means of the characters just pointed out we may always ascertain, even in the most difficult cases, the genetic differences between sarcomata and carcinomata. The first beginnings of plexiform sarcomata and carcinomata are often scarcely distinguishable from each other (compare Fig. 151 with Figs. 176 and 177); in both cases the forms are extremely similar to that of the glands. In the course of their further development, however, a change takes place; the cellular columns of the sarcomata have either had their origin in vessels, or vessels soon grow into them, while this is never the case in carcinomata; but the columns themselves, if they become very

large, remain without vessels, or a cavity forms in them as in the development of glands (compare Fig. 151, *b*, with Fig. 183).

I do not feel called upon to enter further here into a general histological sketch of these tumours, and hope that you will be able to recognise them by what I have said. According to my whole histogenetic conception I cannot admit that an epithelial cancer can form primarily in a bone or lymphatic gland. The observations of this kind known to me (in the under jaw, on the anterior surface of the tibia, in the lymphatic glands of the neck) are, on account of the too great proximity of the skin and mucous membrane, not convincing enough for me; an inconsiderable carcinomatous affection of the skin or mucous membrane might have set in, and have been the starting-point of the disease without having been observed.

The appearance of these tumours in section and their consistence vary so much that nothing general can be said concerning them.

In a very large majority of cases, carcinomata commence as nodules; also as infiltration and induration of otherwise soft tissues, or in the form of exuberant papillary growths. The diseased parts are seldom distinctly separated from the healthy tissues by a capsule of connective tissue; in most cases, the transition from sound to diseased tissue is rather a gradual one. There are cases in which we cannot speak of a cancerous tumour, but only of a cancerous infiltration, because no enlargement but perhaps even a decrease in size of the organ affected is connected with it. It is further characteristic of carcinomata that a portion of the new formation is frequently very short-lived, decays directly or after fatty degeneration, and becomes absorbed, when the infiltrated fibrous tissue contracts to a firm cicatrix. But besides this cicatricial shrinking, and not unfrequently along with it, processes of softening very frequently occur, perhaps still more frequently than shrinking, at all events more extensively. This softening commences for the most part with fatty decay of the cells and caseous metamorphosis; central softening, with bursting outwards and development of an ichorous ulcer, with fungous everted edges, is very characteristic of carcinomata. The mucous metamorphosis of the cell-protoplasm also is a process which sets in in many carcinomata of glands, comparatively most frequently in those of the liver, the stomach, and the rectum. This mucous metamorphosis seldom attacks the stroma of the connective tissue. These mucous cancers are

also called *gelatinous* or *colloid* cancers. If cancerous degenerations become developed on the surface of membranes, the papillary layer may become so greatly increased, the individual papillæ so enormously hypertrophied, that these formations may come essentially to occupy the fore-ground, as in many *papillary cancers* of the mucous membrane of the lips and stomach, &c., and in *villous cancers,* which develop themselves as large papillæ in a dendritic form upon the mucous membrane of the bladder. If the cicatricial shrinking predominates in a carcinoma (as is the case in many cancers of the mammary gland), very hard tumours or indurations form, which have been termed *scirrhus* from an early period. Many carcinomata are *pigmented* brown or black, but *melano-carcinomata* are, on the whole, extremely rare. The majority of soft melanomata are sarcomata.

As regards the *course* of carcinomata, I have already said something when speaking of sarcomata to point out the difference between them; here I will repeat once more that carcinomata always first infect the lymphatic glands situated nearest to them. The infection frequently does not go any further; in other cases, metastatic tumours form in internal organs and in the bones. In the lymphatic glands, however, the small epithelial germs appear to find the most favorable soil for their development. The rapidity of the course varies extremely, of which I shall speak further in connection with the topography of carcinomata. In most cases, no occasional causes for the development of carcinomata are known, in rare cases they have been preceded by injuries and processes of ulceration. What we hear and read of cancer-cachexia and of a specific cachectic appearance in persons suffering from cancer is not confirmed by my observations. Such persons become atrophic eventually like any one who suffers from a serious disturbance of the function of important organs and takes up into his organism products of decomposition from decaying tissues; he becomes anæmic in consequence of hæmorrhages, disturbances of digestion, and deficient nutrition. He then rapidly becomes emaciated and assumes the wax-yellow, brownish, or even brownish-green colour, according to the colour of his skin, met with in other individuals under similar circumstances; I have never observed anything peculiar in such cases. No proof exists that such patients throw out an infectious matter as is believed here and there.

You will now arrive more rapidly at an understanding of the

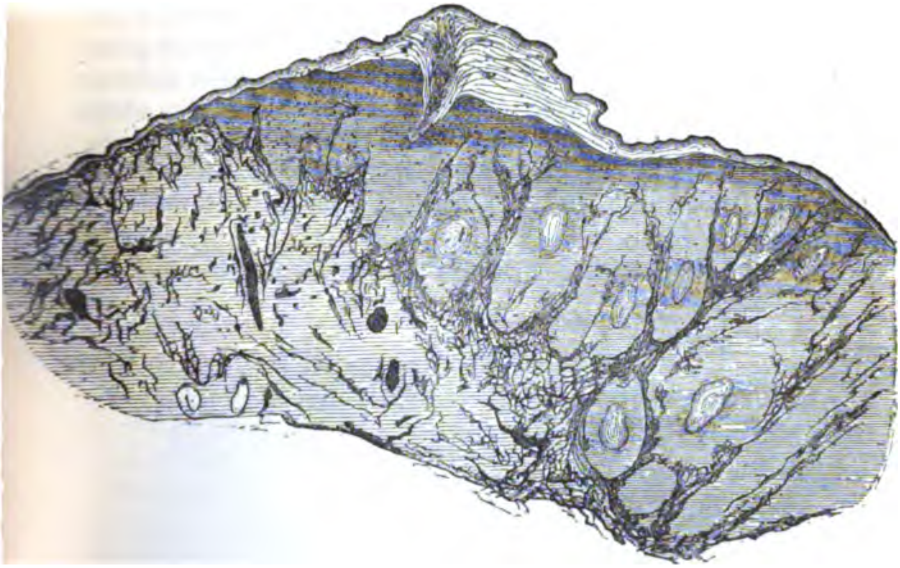
different forms of cancer if we consider them more closely with regard to their origin and to the localities in which they most frequently occur, and, at the same time, enter more fully into the histological details, as well as into the clinical course of carcinomata.

1. *External skin (cutis) and mucous membranes with pavement epithelia. Cancers of the skin. Common epithelial carcinomata* (specially so-called because it was believed of them at first, and, until recently, exclusively, that the chief mass of the cancer-tissue consisted of epithelia), *Cancroid tumours* (tumours resembling cancer; this name was chosen formerly because these cancers of the skin were not regarded as so malignant as those forms of cancer which were observed in the mammary gland and accepted almost exclusively as the type of true cancers).

The cutis is covered with a layer of epithelium, from which, in the fœtus, various prolongations take place into the tissue beneath it, namely, the hair follicles with hairs and sebaceous glands and the sweat glands. In like manner originate the mucous glands in the mucous membranes. It is often asserted that all these structures can form epithelial prolongations, and I will not deny this, but the proof thereof is always most easily furnished for the rete Malpighii. Next to that, a considerable accumulation of epithelia in the sebaceous glands and in the glands of the mucous membrane of the mouth and an enlargement of these glands may most frequently be observed; less frequently, on the other hand, do the hair follicles and sweat glands become implicated. The young cells of the rete retain at first, in spite of these prolongations, their form and size completely; even their relation to the connective tissue of the cutis remains the same, since those cells which lie nearest to the cutis retain the columnar form and direction as upon the normal papillæ of the cutis.

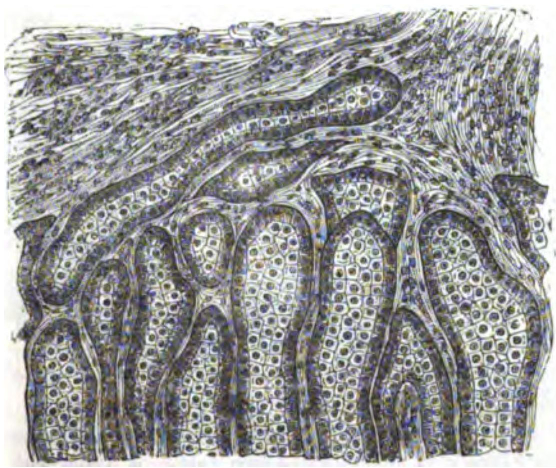
It is very probable that the epithelial, gland-like prolongations not unfrequently grow into spaces between the bundles of connective tissue in which lymph circulates, for the tissue here offers the least resistance. Köster believes that he has proved that all these canals lie in the lymphatic vessels, and in them only. Although his proofs of the correctness of this view are not all sound, there is still something very taking about it, because it would thus be easy to understand why, in these forms of carcinoma, precisely the nearest lymphatic glands sometimes become infected very early.

FIG. 166.



Commencing epithelial cancer of the red edge of the lip, prolongations of the rete Malpighii into the tissue of the lip. Horny scurf. Blood-vessels injected. Magnified 60 times.

FIG. 167.

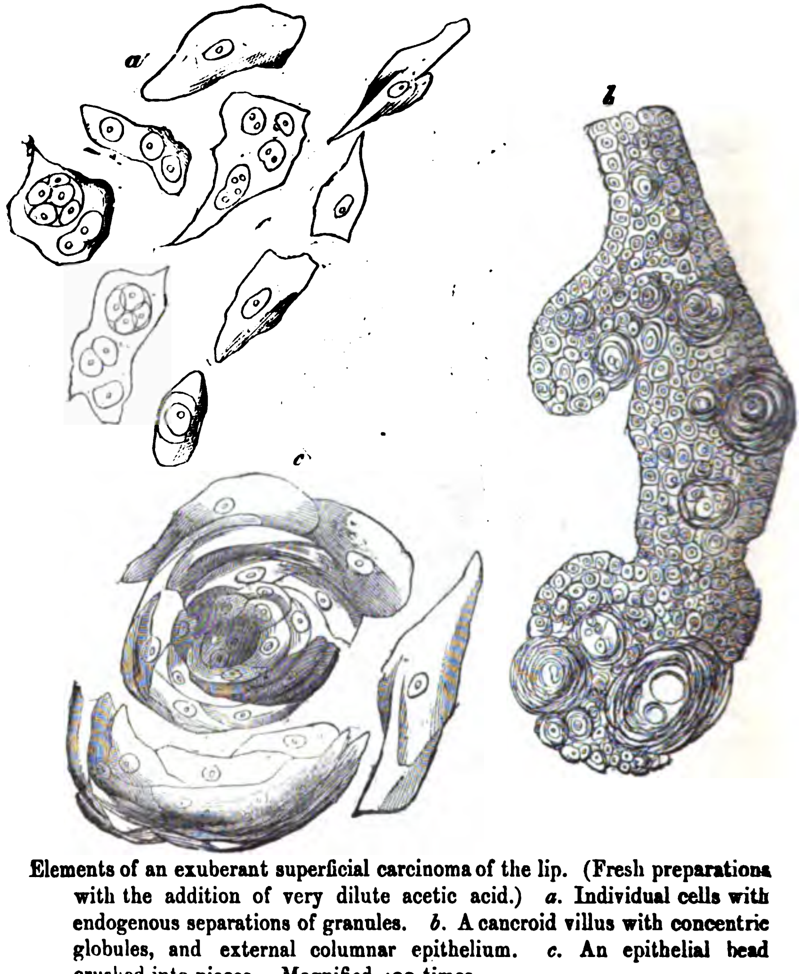


Flat epithelial cancer of the cheek; glandular prolongations of the rete Malpighii into the small-celled infiltrated connective tissue. Magnified 400 times.

In their further course these epithelial canals undergo certain changes; individual groups of cells cohere and form globules, which gradually enlarge by the deposit of fresh cells of the form of pavement epithelium, and thus form the cauliflower epidermis globules (globules épidermiques, Cancroidkugeln, epithelialperlen), which caused the greatest astonishment to the earliest investigators.

It is most probable that these globules, with which we have already become acquainted in sarcomata, where we gave them the

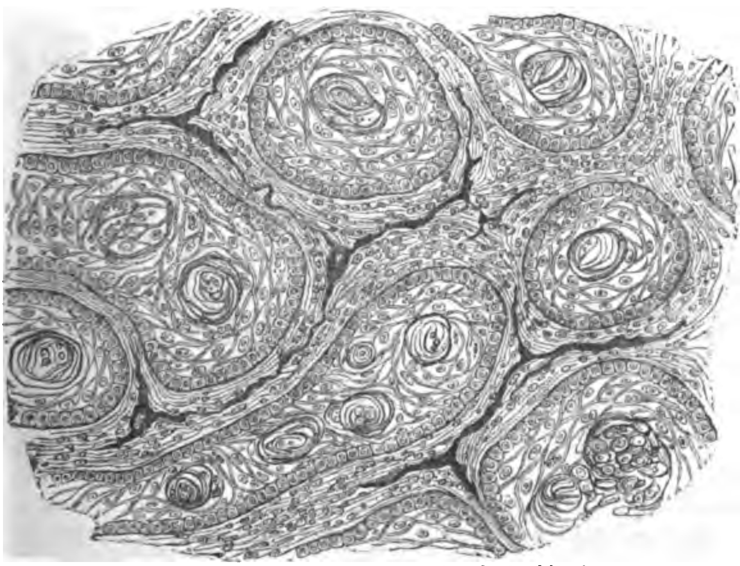
Fig. 168.



Elements of an exuberant superficial carcinoma of the lip. (Fresh preparations with the addition of very dilute acetic acid.) *a.* Individual cells with endogenous separations of granules. *b.* A cancroid villus with concentric globules, and external columnar epithelium. *c.* An epithelial bead crushed into pieces. Magnified 400 times.

name of endothelial globules, result from the separation of a mass of coherent cells into more numerous portions. Amongst the cells in the interior of the globules, as well as in the remaining epithelial parts of these tumours, we often see some with many granules, also large cell corpuscles, which contain younger cells of different ages. If the masses of epithelium have grown deeply into the tissues, and a section is then made in these deeper layers through an indurated tumour of this kind, we observe something like the following appearances (Fig. 169), in which the alveoli filled with epithelium are easily distinguishable from the honey-combed connective tissue.

Fig. 169.



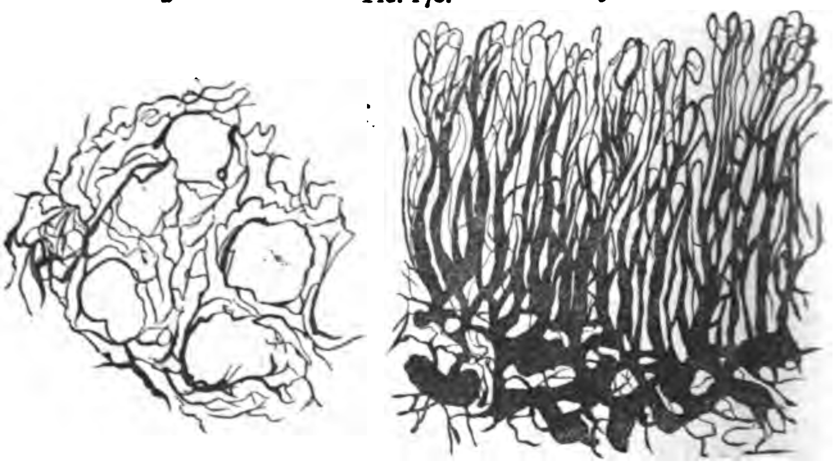
From an exuberant superficial cancer of the hand, the blood-vessels incompletely injected. Magnified 400 times.

The vessels in this connective-tissue stroma appear very much as in Fig. 170 *a*, while Fig. 170 *b* shows an exuberant growth of vessels in enlarged papillæ of a glans penis as it occurred early in the development of the first exuberant epithelial growths.

While, in the last-mentioned case, the papillary hypertrophy shows itself from the first formation of the tumour as an essential characteristic part, in other cases it is altogether of a secondary nature, *i.e.* the epithelial villi on the surface of the skin or mucous membrane soften and fall out and there remains the highly vascular

connective-tissue portion in the form of an indented tumour, from which single papillary tufts project or afterwards grow out. If any one should assert that at the commencement of superficial cancers of the skin, the process always begins with hypertrophy and increased vascularity of the papillæ, to which the correspondingly increasing epithelial cells adhere, and between which they remain firmly fixed, nothing could be objected to this view, in so far as it applies precisely to the beginnings of the formation; for the more deeply penetrating forms of carcinoma which are very rich in cells, this explanation of the relation between papillæ and epithelia would, indeed, no longer suffice.

a FIG. 170. *b*



Vessels from a carcinoma of the penis. Magnified 60 times. *a.* From the fully developed tissue of the tumour, circles of vessels around the epidermis beads. *b.* Loops of vessels from the surface of the greatly indurated, but not yet ulcerated glans penis.

Carcinoma of the skin may commence as indurated papilloma, as a wart, but it begins equally often as a nodule if the exuberant growth remains circumscribed at first, grows into the skin, and now enlarges out of itself without growing larger by the opposition of small fresh nodules of carcinoma. The exuberant carcinomatous growth generally advances from a gradually increasing surface into the cutis, and passes through it, often without causing a very marked elevation.

An essential difference of cancers of the skin from each other consists therein that the exuberant epithelial growth penetrates

sometimes less, sometimes more deeply into the cutis; there are cancers of the skin which remain altogether superficial, scarcely penetrate into the subcutaneous cellular tissue, and grow extremely slowly (*flat epithelial cancers,* Thiersch), others which grow rapidly and penetrate destructively more deeply into the tissues (*infiltrated epithelial cancers,* Thiersch). The above description of cancers of the skin is based chiefly upon the infiltrated form; in flat epithelial cancer the outgrowing cellular columns seldom penetrate further than into the deeper layers of the cutis, and consist principally of the smaller round cells of the rete. Along with these exuberant growths the sebaceous glands increase in size and become filled with fully developed, large-celled epithelia, and the connective tissue becomes infiltrated copiously with small-celled elements. These new formations go on comparatively rarely to the development of epidermis-globules. In this commencing stage, the whole forms, when examined *in sitû,* a hard but little raised infiltration of the cutis, covered with scurfy epidermis. This exuberant epithelial growth possesses very little solidity, however, and occasionally undergoes decay and softening, with expulsion of the sebaceous glands and glandular formations. The highly vascular connective tissue remains behind, and may then go on growing in the form of granulations, or may also partly *become cicatrised.* While this is going on at the centre of the new formation, the latter advances further and further at the periphery, although very slowly.

Cancers of the skin are on section at their very first commencement of a pale red colour and hard consistence, but soon become white and large grained on the section-surface; in the highly exuberant infiltrated forms, we see sometimes the large epithelial beads and large epithelial villi with the naked eye. Ulceration occurs almost more frequently from without inwards than from medullary softening from within outwards, usually pretty soon after the commencement. Mucous softening is very rare in these forms.

As regards the *topography* of these carcinomata, it is to be remarked especially that the following parts of the body are the most frequent seats of these tumours:

(*a*) *The head and neck;* here it is on the eyelids, the conjunctivæ, the skin of the nose and face, the lower lip, the mucous membrane of the mouth, the gums, the cheeks, the tongue, the œsophagus, the larynx, the ear, and the hairy scalp that these tumours most

readily form. The first commencement varies greatly; the worst cases begin at once as nodules in the substance of the mucous membrane or skin, and rapidly go on to ulceration by central softening; other cases begin upon the surface; there is formed a fissure, an indurated excoriation, an epidermoid scurfiness, a soft wart; these diseased conditions, which seem little important at first, may remain for a long time confined to the surface and spread superficially rather than deeply, with moderately indurated edges. If the carcinoma becomes developed from a wart-like formation, it sometimes retains throughout the papillary character. The parts once diseased are destroyed for ever by the metamorphosis into cancer tissue; in very exuberant epithelial carcinomata no cicatricial shrinkings occur. The ulcers which result rapidly from these new formations vary like other cancerous ulcers; the ulcer sometimes becomes gangrenous and forms a crater-shaped gap, sometimes the new formation grows very exuberantly, and ulcers with fungoid overhanging edges form. From these ulcerated surfaces there may not unfrequently be squeezed out a caseous or even puriform pulp, which issues in a grub-like form, similar to the thickened secretion of a dilated gland of the skin (a comedo); this pulp is a mixture of softened epithelial substance and fat. Sometimes earlier, sometimes later, a not unfrequently painful swelling of the nearest lymphatic glands of the neck sets in, which gradually increases; the glandular tumours gradually coalesce, partly with each other, partly with the primary tumour; openings occur at fresh points and the local destruction advances further and further; deeply also, on to the bones of the face or skull, the new formation extends; the bones are destroyed by it, and it takes the place of the bone tissue. Death may ensue from suffocation or starvation in consequence of the pressure of the masses of tumour upon the trachea or œsophagus, or from implication of the brain after perforation of the cranium. It most frequently takes place after gradually increasing marasmus, from complete exhaustion of the powers, with all the phenomena of extreme cachexia. Post-mortem examinations scarcely ever reveal metastatic tumours in internal organs. All these carcinomata upon the head, face, and neck are considerably more frequent in males than in females. The average time during which persons affected with cancer of the mucous membrane of the tongue and mouth survive is one to one and a half years. *Cancers of the tongue are,*

so long as the lymphatic glands remain unaffected, curable, radically, by early and complete extirpation.

In earlier works I have spoken of the form of "flat cancer of the skin," just mentioned, as "cicatrising, shrinking epithelial cancer, or scirrhus cutis," to distinguish it more sharply from the ordinary rapidly growing epithelial cancer. It now appears to me better, however, not to form any such subdivision, and on that account I will state here at once that this flat cancer of the skin is the mildest form of cancer in general, and that, with few exceptions, old people only are attacked by it; the disease sometimes commences as a nodular infiltration of the papillary layer, but always quite superficially; there generally forms first of all an entirely localised accumulation of yellowish-coloured epidermis, a slight scurf, after the removal of which the skin appears at first but slightly reddened and scarcely at all infiltrated; the scurf forms again if it has been removed; after repeated removal, we find beneath it a small, round, dry, papillary, ulcerating surface, having sometimes at this early stage already rather hard, slightly raised edges. This small ulcer, upon which fresh, dry scurf constantly forms, penetrates through the whole thickness of the cutis, but seldom into the subcutaneous cellular tissue; it has a tendency to spread superficially rather than deeply, and it sometimes even heals entirely, as mentioned formerly, at its centre, with formation of a cicatrix and new healthy epidermis, while a slight induration and ulceration spread extremely slowly at the periphery. There are cases in which no ulceration takes place, but only infiltration of the skin, with formation of scurf upon the epidermis and consequent cicatricial shrinking.

The most frequent seat of flat cancer is upon the face, especially upon the cheek, forehead, nose, and eyelids, but other parts of the skin also which are liable, in general, to be attacked by epithelial carcinoma, may be affected by this form. It occurs most frequently between the fiftieth and sixtieth year, and, so far as I know, attacks women just as often as men. The whole surface of the skin, especially of the face and hands, often presents a strikingly dry appearance, with a number of dry, flat, yellowish scurfs upon the epidermis, and at the same time a greater number of infiltrations, which are mostly small and frequently disappear from shrinking. The *spreading* of this cancerous infiltration takes place extremely slowly; from six to eight years sometimes pass away before a piece

of skin of the size of a dollar, or a nostril, an eyelid, or a portion of the ear, &c., becomes destroyed. When this form of cancer occurs in younger individuals, its course is much more rapid and the new formation often passes into the more deeply penetrating form. Since the persons attacked are generally old, they die occasionally of other diseases, and on that account also it often happens that no relapses take place after operations. But also in cases in which no operation has been performed, nor anything whatever done, these forms of carcinoma rarely show themselves to be very infectious; the infection appears never to go beyond infiltrations of the lymphatic glands, which do not occur until late, and then run their course equally slowly and with shrinking, like the primary infiltration. It has, therefore, been proposed to remove this flat form of cancer of the skin altogether from the class of the carcinomata and to place it in that of the chronic inflammatory processes as "ulcus rodens" (Hutchinson), or as a peculiar form of lupus in old people. The frequently occurring combination of this new formation with distinctly characterised cancer in some parts of the infiltrated edges, the possibility of its transition to exuberant cancer of the skin, and several other anatomical and clinical peculiarities, leave no doubt in my mind that this form of infiltration and ulceration is of a cancerous nature, although, as already observed, the mildest and least infectious form in the series of the carcinomatous new formations.

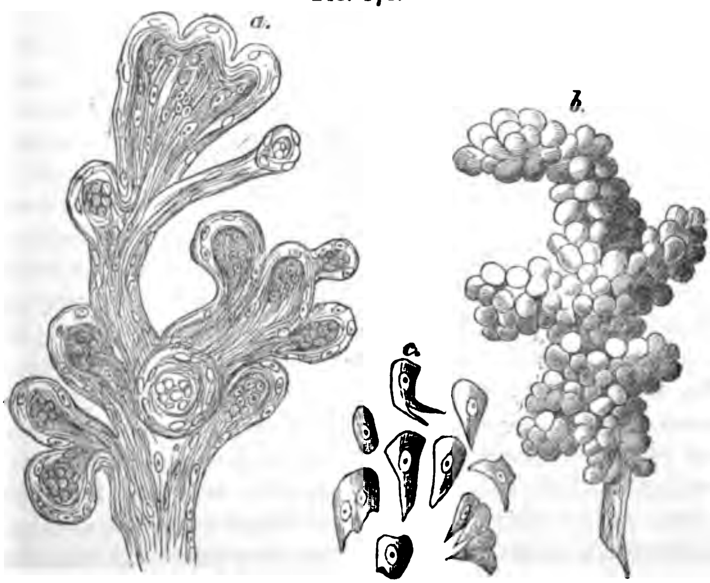
(*b*) *The second part of the body* in which the carcinomata here in question are frequent is the *genitals*. The portio vaginalis uteri, the vagina, the labia minora and clitoris, the penis, and especially the glans and prepuce, are the parts most frequently affected. Of all these parts, the portio vaginalis uteri is that most frequently attacked, and the carcinoma here also passes rapidly into a state of ulceration; and since the surface of the tumour then appears split up at many points, so as to resemble the surface of a head of cauliflower, these cancers have also been termed "cauliflower cancers;" but since sarcomatous papillomata may also cause the same appearance, this term is an uncertain one. At all the points just named the ulcerated tumour may assume at one time rather a destructively ulcerating, at another rather a fungous character, and, moreover, may be either infiltrated or superficial. The secretion of cancer of the uterus is combined with a peculiarly stinking ichor, and frequently with continuous parenchymatous hæmorrhages. As

regards the further course of the disease, the inguinal or retro-peritoneal lymphatic glands become affected sooner or later. Death usually ensues from marasmus, and in these cases also we but *very seldom* find metastatic tumours in internal organs, except in the nearest, directly infected glands.

(*c*) Of other parts of the body possessed of interest surgically, the hand, and especially the back of the hand, must be mentioned. I recently saw a case of epithelial carcinoma on the right arm which had become developed from a fontanelle kept open for ten years by means of peas. I have also seen *an ulcer on the foot* which became cancerous without any known reason after having existed for several years.

(*d*) There now remain to be mentioned here only the carcinomata which commence in the mucous membrane of the bladder, which is also covered with pavement epithelium. Little as these tumours are approachable for surgical treatment, it is still necessary for surgeons to be familiar with them on account of the differential diagnosis. I have already mentioned repeatedly that in carcinoma,

FIG. 171.



Papillary formations from a villous cancer of the bladder, according to Lambl. *a*, Without; *b*, with epithelium; *c*, isolated epithelial cells of the villi. Magnified 350.

exuberant papillary growths occur; this is especially often the case with cancers on the inner surface of the bladder, which frequently grow from it in the form of villi resembling the branches of a tree, whence they have received the special name of "villous cancers."

The cancers which proceed from the chief epithelia and glands stand in the same relation to villous cancers as adenomata to papillomata. As soon as papilloma assumes an especially exuberant character, and masses of epithelium grow at the same time into the respective membrane, so that the connective or muscular tissue becomes infiltrated thereby—in short, as soon as the tumour assumes a distinctly destructive character—it may be regarded as carcinomatous papilloma or villous cancer. The boundaries between a simple papilloma and a villous cancer may sometimes be very difficult to draw.

There forms upon the inner surface of the bladder a tumour projecting into its cavity in the form of algæ and floating in the urine, which at its base stands to the walls of the bladder in the relation of a carcinoma, the frequently very long, tree-like villi of which are covered with large epithelial cells, while the basis of the papillæ consists of connective tissue, in the meshes of which are found epithelial cell-cylinders, as in carcinoma (Fig. 171). The analogy with villous sarcomata is, in fact, very great, except that there (Fig. 149) the villi are covered with endothelia, here with epithelia.

I will add a few words here concerning the general course of the carcinomata of which I have been speaking. They occur, for the most part, at a somewhat advanced age, between the fortieth and sixtieth years of life, very seldom later, but, unfortunately, not unfrequently earlier. I have seen cancer of the tongue in a youth of eighteen, cancer of the uterus in women of twenty. Cancer of the lips appears to occur more frequently in the country than in towns. The earlier these carcinomata occur, the more exuberant not only is, in general, the local tumour, but also the earlier the implication of the lymphatic glands and the more rapid the general course. It has already been observed several times that after complete removal of the tumour no return takes place. In some cases the disease runs its course in a year with great rapidity, in others it goes on for 3—5—10 years and longer (flat cancer of the skin); it also sometimes happens that the return shows itself in the

lymphatic glands *alone*, if, for instance, a cancer of the lip had been *completely* extirpated, but some cancer germs existed at the time of the operation in the lymphatic glands of the neck. The new formation in the lymphatic glands is, at first, of a pale red colour, and in the form of a rather hard, diffused infiltration or of a white node, but becomes softer in the course of time, and partly pulpy and suppurative. The cancerously infiltrated lymphatic glands of the neck have a great tendency to ulceration. The microscopical structure of the infected glands resembles that of primary cancers. I consider it established beyond all doubt that secondary cancer in the lymph-glands results from transplantation of cancer-germs from the primary seat of disease. The above described varieties of cancer seldom get beyond the lymphatic glands; infection of internal organs (liver, lung, spleen, kidneys) are decided rarities. The frequency with which carcinoma appears at certain parts, such as the transitions of mucous membrane into skin (vagina, penis, lips), has always and very properly roused special attention. It is natural to seek for the causes of this disease either in the structure of the parts, or in the irritations to which the openings are exposed. In consequence of the dislike which most modern pathologists evince towards specific, unknown irritations, first one and then another cause has been brought forward with a view to clear up the obscurity of the formation of specific tumours. Thiersch, in regard to the lips of elderly persons, attaches weight to the fact that great changes take place in them, and especially in the skin, in the course of years; considerable atrophy of the connective tissue and of the muscular structures takes place, so that the epidermic structures, such as the hair follicles, sebaceous glands, sweat-glands, mucous glands, freed from a certain amount of pressure, come prominently forward and receive a larger supply of the nourishment in comparison with the connective tissues, and hence all irritants which react on the lips, such as bad shaving, smoking, wind and weather, tend to excite a reactive proliferation in the glandular parts of the lips, and to set up a hyperplastic activity in these structures. In England epithelial cancer is fairly common in the scrotum among chimney-sweepers (chimney-sweepers' cancer), and is believed to be due to the irritation of the soot. These influences may certainly help, but they do not altogether explain why cancers—infectious diseases—always result, rather than chronic inflamma-

tions, catarrhal affections, or the like. I will not further enter on this subject here, but must refer you to what has already been said on the ætiology of cancer-formation in the introductory chapter on tumours.

2. *Mammary glands.*—I introduce cancer of the breast in this place because these glands are also derivatives from the epidermis; they are skin fat-glands on a large scale. Nevertheless, the varieties of mammary cancer deviate somewhat from the already described skin cancers; and even though true epithelioma of this gland setting out from skin of the nipple does sometimes occur, it is exceedingly rare. Mammary cancer, unfortunately a very common disease, begins, it appears to me, almost always coincidentally with an increase of the small round epithelial cells in the acini, and with a small cell infiltration of the surrounding connective tissue.

It is, as I have already remarked, scarcely possible with our present methods of examination to make out whether the *first* changes take place in the gland-cells or in the connective tissue; for the heaping up of small round cells *around* the acini soon becomes so excessive that it is quite impossible to follow out the further changes *within* the acini. As the result, however, of a fairly extensive experience in this subject, I think I am justified in giving the following as the subsequent course of the process.

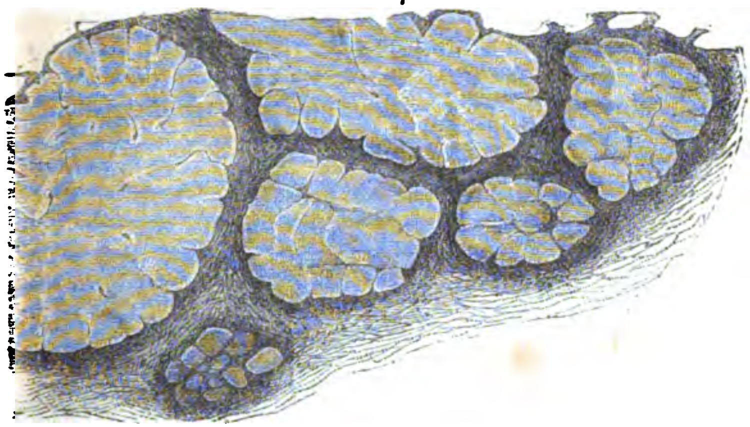
The heaping up of cells within the acini leads, first of all, to their enlargement, and sometimes to the discharge of a very small quantity of secretion from them (as is demonstrated by the outflow of serum from the nipple).

In consequence of continuous cell proliferation a still further dilatation of the acini takes place in such different ways that (1) an acinous and generally large-celled, and (2) a tubular and, for the most part, small-celled form of mammary cancer can be distinguished. The former leads to the development of large lobulated glandular nodules; I give you the name, therefore, of the "acinous form," because the form of the glandular acini are preserved, although only in their rougher outlines. A low magnifying power would give the following representation of such a tumour (Fig. 172, p. 473).

The groups of epithelial cells, enlarged and converted into thick gland clusters, are surrounded by infiltrated connective tissue, and are also permeated by a fine network (stroma) of the same tissue. This I consider to be the remains of the firm sheath-wall existing between the acini, but which, however, is considered by other

authors to be newly formed tissue. If a section be made through a well-hardened specimen of soft acinous mammary cancer, the

FIG. 172.



Cancer of the breast. Acinous form. Magnified 50 times.

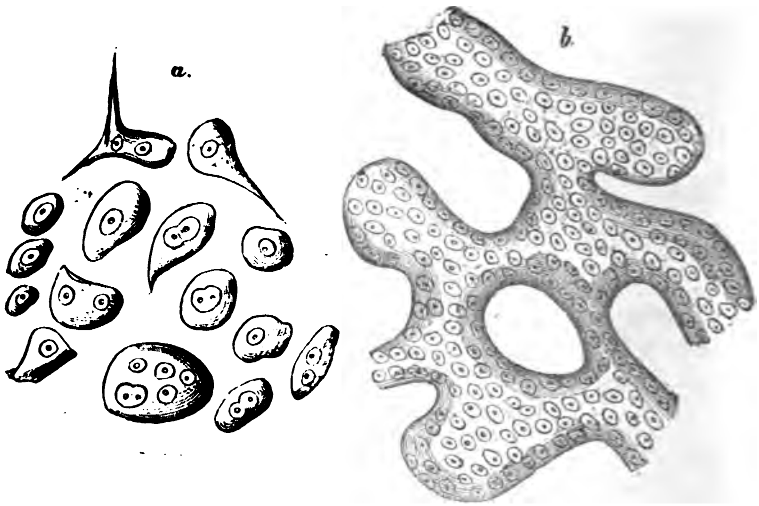
inter-acinous tissue, under a moderate magnifying power, would appear as in Fig. 173. I consider all the cells in the large meshes of the connective tissue to be of epithelial origin.

FIG. 173.

Soft mammary cancer. Alveolated tissue of the carcinoma. Spirit preparation. Magnified 100 times.

This variety of mammary cancer is generally soft; on section it is granular and grey-white (medullary). If we scrape the cut surface of such a tumour with a knife, by means of which we shall get a thick whitish juice, and examine it fresh under the microscope, we find gland-shaped clusters of cells, very pale, and made up of variously shaped cells with large nuclei. Many of these cells contain several nuclei, some of them are in the act of subdividing.

FIG. 174.



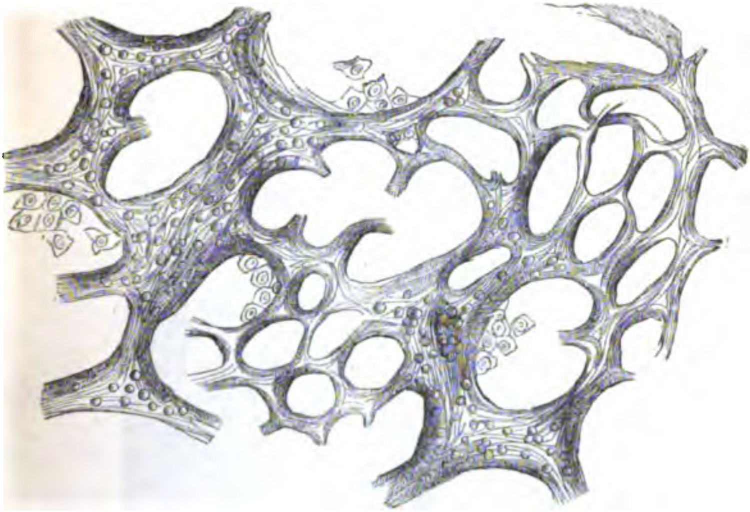
From a cancer of the breast. *a*. Cells with many nuclei (fresh preparation with a drop of water added). *b*. Gland-like cell-cylinders (fresh preparation). Magnified 300 times.

The connective-tissue stroma in which these elements are lodged, when washed, appears as in the next drawing under a medium magnifying power (Fig. 175).

The next most frequent variety (which is harder and on section pale red) may be termed the tubular form of cancer, because in it the acini do not retain their shape and form, but *grow into* the connective tissue as long cellular cylinders; the connective tissue at the same time becomes infiltrated with small cells. As the cells which have proliferated from the epithelium in this form of cancer do not attain to the same size as those of the previously-described variety, and as the cells which are found in the connective tissue are heaped up very closely, it is obvious how difficult it must be to distinguish the cells which are derived from the glandular epithelium

and those which belong to the connective tissue, and which are really wandering cells.

FIG. 175.



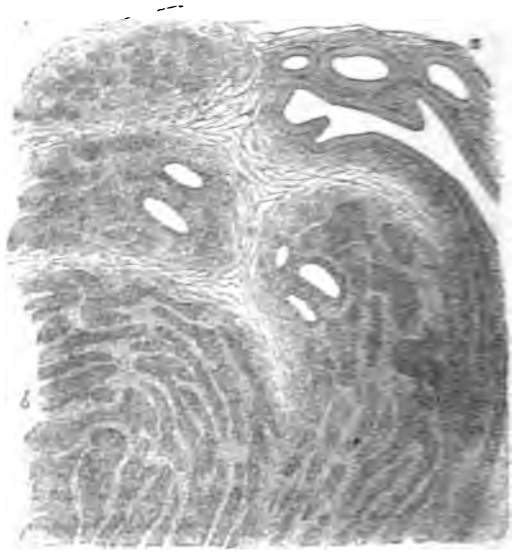
The conuective-tissue-stroma of a cancer of the breast; the thicker strands are much infiltrated by young cells. Alcohol preparation brushed out. Magnified 100 times.

Thus, then, authorities are not all yet agreed that all these common forms of carcinoma of the breast are really cancers; for some believe that all the cells found in these growths proceed from the connective tissue. The developmental history of these growths can only decide this point; but at present as we do not possess any means of distinguishing the young derivatives of epithelium under all conditions from wandering colourless blood-cells and from the derivatives of the connective-tissue-cells, so long will it be impossible to say whether this form of mammary cancer partakes more of an epithelial or of a connective-tissue type.

Although all forms of cancer of the breast show a tendency to ulceration, it is, nevertheless, chiefly the case in the softer forms of cancer. The hardness of a mammary cancer, however, does not always determine the amount of its cellular elements, for acinous forms of cancer, rich in cells, may, nevertheless, be hard, if these cells, as in the normal acini, are enclosed in a small tightly packed

capsule of connective tissue. Softening takes place centrally in the nodule lying near to the skin, or it may occur in a hard cancer from without inwards at places where the tumour is adherent to the skin. Mucoid degeneration of the stroma or of the gland-cells

Fig. 176.



Mammary glandular cancer—tubular variety. Magnified 150 times.

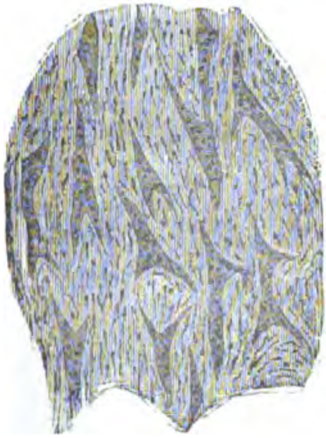
occurs but rarely: Doutrelepont has recently described such a case. The softening patches appear to the naked eye yellowish-white and granular (caseous or fatty softening), or, in very vascular growths, greyish-red or dark red, especially if extravasation of blood have taken place. Cysts occasionally are formed in consequence of these softening processes and the encapsuling of the products. Retention and secretion cysts may also form either in the gland structure or in the cancerous nodule itself.

Cicatricial processes are very common in mammary cancers; the nipple and other parts may thus be cicatricially retracted.

The microscopic examination of these shrunken parts shows strands of connective tissue with shrunken connective-tissue-corpuscles, and sections of small branched canals (shrunken alveoli, Fig. 177) filled with cellular *débris* or fat. This shrinking of the new growth, in many cancers of the breast, is such an important feature, that a special variety of cancer has, on this account, been named

"shrinking or cicatricial cancer." It cannot be denied that this form of cancer does present peculiarities, which distinguish it from

FIG. 177.



Cancer of the breast, taken from a well-shrunken part. Magnified 200 times.

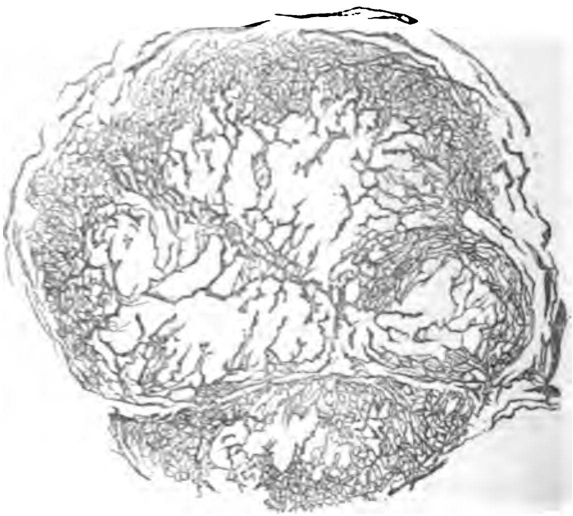
the common and more frequent varieties of cancer. I prefer, therefore, to speak more specially of it later on.

The development of cancer is associated with a not inconsiderable development of vessels, and also of vascular dilatation. In the neighbourhood of the more recent parts of the new growth a very rich supply of fine vessels and vascular loops (Fig. 178) is found, and in the older and softening parts the vessels are at first widened (Fig. 179), then become thrombosed, and finally perish; thus, then, around the softening portions of a tumour vascular loops are formed similar to those which take place during the formation of an abscess.

I will make a few remarks on the clinical symptoms which accompany the development and course of an ordinary case of cancer of the breast. The disease, as a rule, commences between the thirtieth and sixtieth years of life—seldom earlier, seldom later. The affected woman is with this exception, as a rule, perfectly healthy; sometimes even of robust appearance, fat, and in good condition; married and unmarried women, those having had children and those without, and of all stations in life, are attacked. Not infrequently the parents or grandparents have died of cancer. Most commonly a small painless tumour forms in one breast, generally in its under

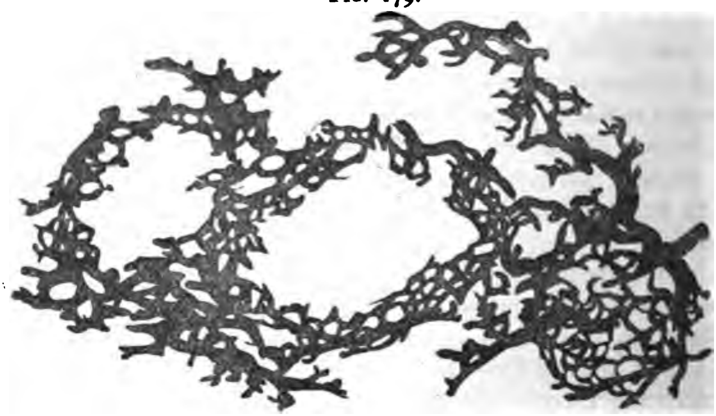
and outer part; this may remain unobserved for months; it is of a hard consistence, and lies infiltrated in the gland, but at first

FIG. 178.



Vascular loops around a quite recent nodule of mammary cancer. Magnified 50 diameters.

FIG. 179.



Vascular loops around a softening spot in a mammary cancer. Magnified 50 diameters.

movable beneath the skin and on the pectoral muscles. Its growth at first is moderately fast; possibly a year elapses before the tumour gets to the size of a small apple. The size of these tumours is

not always alike; sometimes the tumour is large and tender, especially before and during the occurrence of the menses; sometimes a tumour grows more rapidly during pregnancy; sometimes, however, it decreases in size and becomes stationary. These symptoms for the most part are due to congestions of the mammary glands or to cicatrisation and wasting in the tumour. In due time, that is in the course of months, the tumour grows more rapidly. The skin over it becomes fixed, and adhesion with the pectoral muscle takes place in the deeper parts. The patients seldom notice the earliest implication of the lymphatic glands in the axilla, and were it not for the fact that a medical examination of the axilla is made from time to time, the formation of tumours within the lymph-glands (which shows itself at first as a hard swelling) would only come under observation very late on in the disease. These glands lie either so high up in the axilla, or so low down beneath the pectoralis muscle, that they can only be felt after they have attained a certain size.

The lymphatic glands of the neck are rarely affected in mammary cancer; if they should become affected the prognosis becomes much more unfavorable. If now the tumour is allowed to develop without any hindrance, the result of cases where there is a moderately slow growth is about as follows:—The tumours in the breast and in the axillary glands gradually become confluent, so that a nodulated, roundish, immovable mass results, which in some places is adherent to the skin; the pressure of the tumour on the nerves and vessels in the axilla causes neuralgic pains and œdema in the arm; the patients who up till this time have been quite well, in consequence of the pain and swelling of the arm (which are much worse at night than in the day), are now compelled to keep their beds; the pains are of a shooting and gnawing character. Another symptom usually appears at this stage (we are supposing that about two years have elapsed since the first appearance), viz. ulceration. This usually appears with the following symptoms: A portion of the tumour grows out into a nodular form, the skin, which is getting constantly thinner, at last becomes red and its vessels dilated; finally a rent or blister appears on the raised, reddened, and almost fluctuating apex of this portion of the tumour; next that portion of the cancer-mass which is exposed to the air becomes gangrenous and sloughs off in bits; a deep crater-like ulcer forms, which remains so for a long time if the surrounding structures are hard; but

if they, on the contrary, are soft, then the tumour begins to proliferate at its edges and from its deeper portions, and the granulations after a while overlap, like a mushroom, the margins of the tumour. In this manner an ulcer forms, sometimes with torpid, sometimes with a fungating character; the secretion from it is a foul, stinking serosity, which contains also shreds of sloughing tissue. But what is worse, hæmorrhages, generally parenchymatous, though sometimes also arterial, occur, and consume the patient's strength. We have now followed the condition of our patient until she has become almost or entirely bedridden, and we next come to the catastrophe. The patients become pale and emaciated, the appetite is destroyed, their strength fails, their nights are sleepless, because the pains increase in intensity; opiates have to be given freely in order to obtain a little rest and sleep and mitigation of their sufferings. Now we have a complete picture of the so-called cancer-dyscrasia or cancer-cachexia before us, and of which we have already spoken. Months may pass; the fœtor from the tumour poisons the room; the patient becomes weaker, the skin of a yellowish earthy colour, then pain on inspiration and in the region of the liver, and also in the extremities, supervenes; finally the patients die of marasmus after a long, lingering, and painful agony; sometimes pleurisy or peritonitis happily hastens the result. On making an autopsy, we generally find carcinomatous tumours in the pleura and liver, sometimes also on the ribs corresponding to the tumour. The disease in its entirety has lasted about two and a half years.

This sketch will answer for very many cancers of the breast, yet there are various modifications in the course of these cases. First, the rapidity of the local manifestation varies much; the tumour may remain confined to the breast for years without affecting the lymphatics; this is an exceedingly rare variety. Again, the disease in the glands may show itself simultaneously almost with that in the breast; this always pre-indicates a rapid course for the disease, while the opposite—a late and only moderate affection of the lymphatics—generally indicates a milder and slower course for the whole disease.

Carcinoma may occur simultaneously in both breasts, or in one very shortly after the other; prognosis becomes less and less favorable under such circumstances.

In some cases no isolated tumour occurs in the breast, but the

whole gland, together with the skin, seems to become diseased all at once. The occurrence of small nodules over the skin gradually spreading to the surrounding parts is a serious matter from a prognostic point of view. In such cases the course is usually a short one, even although there may be no ulceration and the tumour a hard one.

Finally, an adenoma or a sarcoma of 8—10—15 years' growth may suddenly assume the character of a cancer, that is, it may become fixed, immovable, and painful, and the lymphatic glands may commence to indurate.

Cases occur in which the mammary tumour so contracts and shrivels that one is inclined to believe that it has entirely disappeared; unfortunately, this does not prevent the general outbreak of carcinosis, although it may postpone it for awhile; or it only occurs in the milder cases, which run their course in from four to six years. Many patients die early from ulceration and hæmorrhage or from metastatic growths. As regards the period at which metastatic growths appear in the internal organs, this varies very considerably; in general it seems as if metastatic growths occur much later in those cases where the local growth of the tumour is a slow one; there are exceptions to this rule, however. The localisation of local tumours is remarkably uniform in mammary cancers; as before said, the pleura, liver, bones (humerus and femur), are the most frequent seats of metastatic tumours. The variable course of mammary cancers makes it very difficult, almost impossible, indeed, to compare the result of early or of late operations with those cases which are allowed to go on without any operation at all. The age alone makes a great difference; the disease always runs a slower course in old than in younger individuals, and besides this a number of unknown causes comes into operation. The most opposite rules for treatment and operation have been laid down by the most experienced surgeons; the one believing that the course of the disease would be cut short by an operation, while another fancied that it would be accelerated. The statistical tables, which are at present available, in no way help us to decide this important question, because all kinds of cases are thrown together; in order to arrive at any definite results the cases ought to be carefully arranged according to some given principles. And yet how could this help us? In each individual case the question is, can we or can we not relieve the patient by the operation? The tumours almost always recur either in the scar itself or in its vicinity, or in the lymphatic

glands, because the cases generally come under treatment too late. So the patients die either from the suppuration, or hæmorrhage, or of acute diseases, or of metastatic outbreaks elsewhere. Unfortunately, the prognosis, save in a few exceptional cases, is easily arrived at. Does the patient suffer much from the tumour? Is there any local danger? These are the most important questions. I am alluding here to therapeutic measures, although I purpose to enter on the subject more fully at the latter part of the section on cancer. An examination of the enlarged and partly adherent lymph-glands shows them to be more juicy and more vascular than normal; the larger ones contain hard white or greyish-white nodules, while the largest are sometimes breaking down, caseous, and have a granular look when cut into. On the whole, the lymph-glands present the same characters as the primary cancers, even to the microscopic appearances. Although it is only in pigmented cancers that it can be shown that the first swelling of the lymphatic glands is due to the transference of cancer elements directly into the glands, I nevertheless consider it the same for all cancers. In some cases the epithelial nature of the new growth in the lymph glands is just as obvious as in the primary cancerous nodule, yet in other cases such a differentiation is not possible. As regards the direct transference of cancer nodules on to the pleura in cases of mammary cancer, it may be said that they are mostly hard, pure white, and small celled; the same may be said of the external characters of secondary growths in the lungs or liver. The latter, however, are not infrequently large celled and acinous. However probable it may seem that these cancers also result from the direct wandering of cancer cells or from a transference of them through the blood or lymphatic vessels, it is not yet proved to be the case.

Some cases do not conform to the above given description, but are characterised by an early and continuous shrinking of the new growth. This form of cancer of the breast to which I have already briefly alluded, is called atrophic, or shrinking cancer, or connective-tissue cancer. The nature of the disease and of the pathological process may be inferred from what here follows. A hard lump appears in the breast, seldom before the fiftieth year; it cannot be called a swelling, for along with the induration there is rather a partial or even a total diminution in the size of the gland; this induration generally takes place without any pain, and seldom is

the pain, if any, very severe; it is extremely slow in its growth, generally going on for years and years. If we imagine the diseased gland removed at this stage and examined it, we should find a

FIG. 180.

Infiltration of the connective tissue on the borders of a cancerous nodule of the breast, spreading to the skin. The shaded parts represent the advancing small-cell infiltration. Magnified 50 diameters.

tissue of such toughness that we can scarcely cut with a knife; the cut surface presents to the naked eye a very dense fibrous-looking cicatrix, with strands of connective tissue radiating in all directions into the normal tissues around. In a well marked example of this form of cancer scarcely any other pathological peculiarity can be detected; in some of these tumours, however, about the peripheral parts, more marked in some places than in others, may be seen a pale reddish-shiny, albuminoid part, which is situated between the tumour and the healthy structure, and which runs into both.

If these sections, especially of the central parts, after having been previously hardened in spirit, be examined under the microscope, one finds nothing but connective tissue with elastic fibres; the fibres, however, have not the same regular course as in a fibroma, but run irregularly among themselves and, as already remarked, are mixed up with elastic fibres, which is very unusual in fibromata. Examination of the peripheral tissue gives a different result; here there is cell infiltration, but in very moderate quantity;

small groups of pale lymph-like cells with single nuclei are developed, as in the commencement of other neoplasia. A portion of these cells is arranged in elongated groups (tubules), some are larger than others; these are no doubt derivatives of the remains of the epithelial cells of the atrophied gland acini. All the cells of this new growth, however, seem to be very short lived, for they are scarcely formed before they begin to waste, without any attempt at further development; then the connective tissue in which they are enclosed, somewhat open and lose its structure, begins to contract and become close and compact, and as a result of this process we get a cicatrix.

But as this small-cell infiltration—though small in quantity —spreads persistently towards the periphery, it never or *very* rarely, happens that a complete, spontaneous, cicatricial healing up of the cancer takes place. On examining sections taken from the peripheral portions of these tumours under a low magnifying power, it is seen that the small-cell infiltration makes its way between and among the meshes of the connective tissue, which it closely follows.

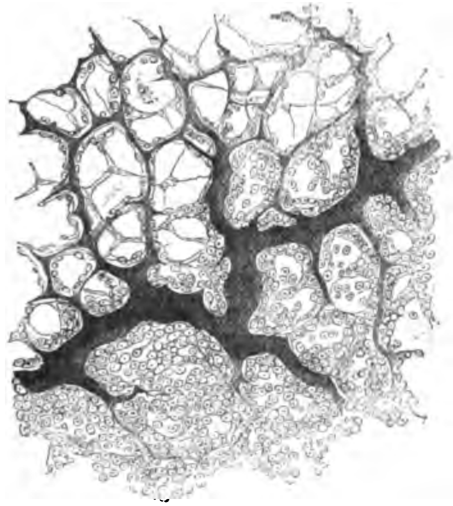
The spread of this infiltration into the connective tissue is exactly the same as in inflammation; the majority of young cells is found in the neighbourhood of the vessels, so that one can hardly avoid the conclusion that, in these cases, they are the colourless corpuscles of the blood wandering out from the blood-vessels, and so giving rise to the cell infiltration.

As the infiltration of the connective tissue with lymphoid cells becomes, very obviously, the most prominent part in this diseased process, and as the epithelial proliferation falls quite into the background, I formerly proposed and endeavoured to introduce the name of "connective-tissue cancer" for this form of mammary carcinoma. But as this appelation might possibly lead to a misunderstanding as regards the modern anatomical definition of the term cancer, I have not insisted on its continued use.

The above-mentioned peculiar anatomical and clinical course has induced some surgeons to desire that this neoplasia should be scratched out of the list of tumours altogether, certainly from the list of cancers. Let us consider the clinical course of these cases a little more closely: we have already remarked that, as a rule, only elderly individuals are attacked by this disease, and that the local affection is extraordinarily slow; some cases last for seven or eight years, until the half of a breast has completely atrophied.

The general health is meanwhile quite undisturbed. The lymph-glands sometimes take part in the disease, and the disease goes on

FIG. 181.



Cell infiltration of the adipose tissue in the periphery of a hard mammary cancer. The blood-vessels are injected. Magnified 200 diameters.

in them exactly as in the mammary gland itself; there is very little enlargement, though there is great induration and cicatricial atrophy. The more quickly and the more thoroughly the new growth goes on to atrophy the slower does the disease spread and the less dangerous is it. Local recurrence after extirpation of, or caustic applications to, this form of cancer does not often take place, or it occurs very late. Metastatic growths scarcely ever occur. Pathologically this kind of infiltration appears not to differ essentially from that seen in chronic hepatitis and nephritis with consecutive atrophy, and thus why separate this form of scirrhus from those processes? Wernher has described this disease of the mammary gland as "cirrhosis mammæ." I willingly acknowledge the justice of doubting the carcinomatous nature of many cases of scirrhus of the breast, but on the whole prefer to reckon them among the cancers, and for the following reasons:—the process of shrinking, as you already know, is peculiar to the cancers; but it must be especially remarked that these shrinking cancers are not infrequently associated with the ordinary form of carcinoma; it is, indeed, the

more frequent condition to find a more or less extensive carcinomatous proliferation side by side with the cirrhosis mammæ, while the unmixed form of a completely atrophying cancer is relatively rare. These combinations, which are not met with either in cirrhosis of the liver or of the kidney, speak strongly in favour of a close relationship between this cicatrising neoplasia and cancer, and furthermore, in these combined cases, local recurrences after extirpations, affection of the lymphatic glands, and even metastatic cancers in internal organs, do occur. In the tumours, which consist *chiefly* of cicatrising tissue, and which, therefore, belong to scirrhus rather than to the ordinary form of cancer, a relatively favourable prognosis can be given, because the disease runs such a slow and chronic course.

Further, we must mention another form of cancer of the breast, which also begins as an induration in the gland, but which very soon spreads to the skin, and then, in the form of small nodules, rapidly extends over the whole surface of the chest-wall; the opposite breast may become affected in like manner. This form of cancer (cancer lenticularis, Schuh; Squirrhe pustuleux ou disséminé, Velpeau), occurs either as a primary form or as a recurrent form after extirpation of cancers of the breast, and, as a rule, in not very old women. This small nodulated (one might almost say tubercle-like) form of cancer, in consequence of confluence and of shrinking, may advance until the integument of the thorax, both in front and at the sides, literally constricts the chest (cancer en cuirasse, Velpeau). The course is usually a slow one, the tendency to metastases in internal organs is not very pronounced, but the prognosis is nevertheless bad, because every attempt to prevent local spread by operation is in vain.

3. *Mucous membrane with cylindrical epithelium.*—Most of the cancers which develop in the nose and in the antrum of Highmore, and which gradually spread to the upper jaw, to the ethmoid and sphenoid, also into the orbits, proceed from the mucous membranes of the nose and of the antrum of Highmore.

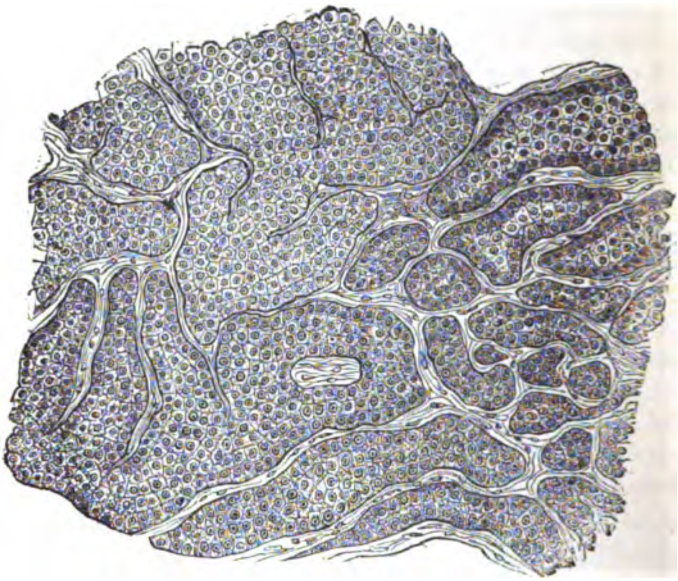
The ciliated and the non-ciliated cylindrical epithelium of these membranes only reach as far as the orifices of the mucous glands, and but rarely go very deep even on the development of glandular cancers in these parts. It appears to be the glandular acini themselves from which the proliferation starts, for these cancers consist,

for the most part, of acini and tubuli, which are lined by, smaller or larger, round cells, rarely by cylindrical cells, and more rarely still by ciliated epithelium. The shape and size of the newly formed acini vary immensely, yet they are often so well marked, so normal, that they may easily be mistaken for clusters of normal mucous glands; and to make this deception more complete we not infrequently find that the newly formed acini secrete a mucus, which remains enclosed within them. When the secretion is retained, the shape of the newly-formed gland clusters not exactly round, and the interstitial connective tissue only moderately developed, fine sections of the hardened preparations of such a tumour present a striking likeness to the tissue of the thyroid gland. The interstitial tissue in these tumours is mostly very soft, as in the corresponding mucous membranes themselves; it may be almost mucoid. These tumours may well be mistaken for plexiform sarcomata and cylindromata, and, indeed, this cannot always be avoided.

The consistence of these tumours is always very soft, their appearance white, medullary, and gelatinous; only when the tumours are very vascular do they seem to be dark red. Bones are attacked, as in caries, without the slightest evidence of any osteoplastic reaction and without osteophytes. These tumours present much that is peculiar in their outward characters and in their clinical features, differing much from other cancers. They occur from about the twentieth year of life up to almost any age; they grow rather rapidly, and soon present either through the nares, or through the cheek, or at the inner angle of the eye; they are sometimes remarkably well-defined and sharply encapsuled, as may easily be made out either by palpation or at the operation; sometimes when situated in the upper jaw they are more diffuse. As yet, I have never seen any affection of the lymphatic glands in these cases of cancer of the mucous glands of the face, and I feel persuaded that these patients may be completely cured by an early and thorough operation. In all the patients on whom I have myself operated I have never been really sure that complete removal of the cancerous masses has been accomplished at the operation; indeed, I have always found that these tumours have grown backwards or so far upwards that any radical operation would itself have been directly dangerous to life. Thus, I have generally seen these tumours recur locally; they kill either by marasmus or pressure on the brain, or the patients die in consequence of too extensive operation.

In none of my own cases have I seen any secondary internal tumours.

FIG. 182.



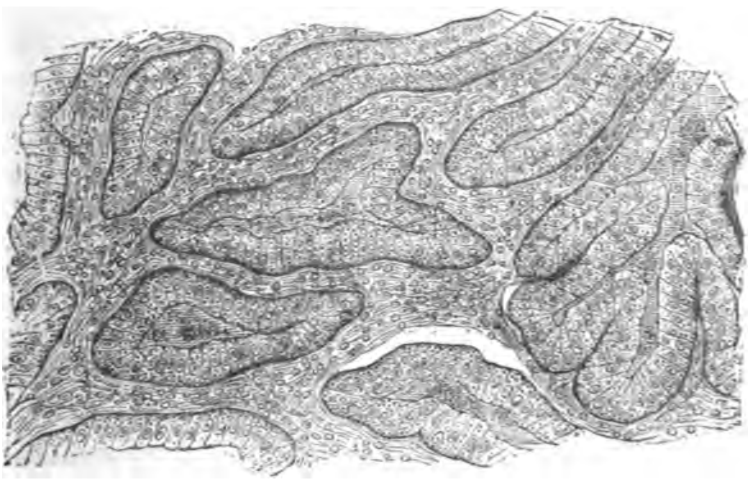
Cancer from the interior of the nose. Magnified 200 diameters.

Glandular cancers often occur in the stomach, especially with mucoid degeneration (gelatinous cancer) and secondary cancer of the liver. Cancer is very rare in the duodenum. As regards cancer of the intestinal tract, we, as surgeons, are only interested in cancer of the rectum. In these cases the growth proceeds almost exclusively from the glands of the large intestine, which grow out in the form of elongated, and sometimes branched, saccules; the lumina of the glands are often preserved and filled with mucus; the cylindrical epithelium retains its form and becomes very large. The interstitial connective tissue becomes infiltrated with small round cells, it undergoes a partial mucoid softening, and is very vascular. At the commencement of the disease the muscular coat of the gut becomes hypertrophied, but later on it becomes involved in the ulceration which early comes on.

As the earliest symptoms of cancer of the rectum are constipation, the passage of mucus with the stools, and slight hæmorrhage, such patients are often treated for a long time for hæmorrhoids before a

manual examination leads to a correct diagnosis. Induration and nodular infiltration, prominent granulations commencing just above the sphincter ani muscle, and spreading around the entire circumference of the gut, lead to a stricture in the form of a thick, padded ring, of greater or less extent. The removal of this new growth can only be accomplished by extirpating the entire rectum. When the extirpated rectum is examined a wall-like, raised ulcer with prominent edges and an indurated base is seen; the surrounding tissue is much infiltrated, and in many places also there are evidences of contraction. The inguinal and retroperitoneal glands become affected, but late on in the disease. The patients generally die in consequence of intestinal stenosis, or of marasmus, or of hæmorrhage and local sloughing.

FIG. 183.



Cancer of the rectum. Magnified 200 diameters.

Cancers with typical cylindrical epithelium also sometimes start from the glands in the pars cervicalis uteri; they then grow into the uterus, and little by little the whole neighbourhood and the retroperitoneal glands become affected and infiltrated; they merge into the epithelial cancers (with flattened cells) and present no great differences in their further course.

4. *Salivary glands and prostate.*—The salivary glands may be the seat of cancer, but it does not occur until late in life; then it grows rapidly, and appears not infrequently with symptoms of

chronic inflammation. The appearance of the newly formed acini is often more tubular than acinous. Epithelial "nests" (pearls) occur at the extremities of the tubuli, which are lined with cylindrical cells. These patients usually die of general marasmus; metastatic internal cancers are exceedingly uncommon in these cases.

I have seen a few times cancer of the prostate in old people; it was very soft, and in one partially extirpated case very vascular and of an acinous structure. From the excellent statistical record of malignant tumours of the prostate by O. Wyss, it may be seen that this form of carcinoma almost always kills by its local effects. Lymph-glands and neighbouring parts may be affected, but secondary deposits in internal organs are seldom found.

5. *Thyroid gland, and ovary.*—I place these two organs together because they are each formed from true glandular epithelium, and both contain follicles derived from a constriction of glandular tubules. Both organs when affected by cancer revert back to the embryonal type, that is, the follicles again grow into tubes and crypts, from which eventually new follicles are further developed. Sometimes however, some of these, on the whole rather uncommon, forms of cancer consist entirely of cellular cylinders, without formation of follicles. Young persons, as well as older ones, may be attacked by this form of cancer; their course is generally rather rapid, as the cancer, when it affects the thyroid gland, usually invades the windpipe, or by pressure occludes it; when it attacks the ovaries, in consequence of its rapid growth and development and extensive implication of surrounding structures, it quickly leads to dangerous ascites.

On account of the many differences, both in the clinical course and anatomical structure of the various forms of cancer, we have had to divide them into groups; we can, however, speak of treatment under one heading. It is usual to describe the treatment of the carcinomatous dyscrasia (carcinosis) as the "*partie honteuse*" of Medicine and Surgery. I cannot altogether subscribe to such a view. It is true we cannot cure the disease; but is it not also the case with many other acute and chronic diseases? Can we stop a catarrh at any stage of its course? Can we arrest the course of one of the acute exanthemata, or of typhoid fever? Can we cure tuberculosis? Certainly not; in all these cases we can do little with medicine, and least of all do we adopt heroic measures. In

carcinosis our therapeutic measures appear to us so unavailing, chiefly because the disease is almost always fatal, and because we cannot in any way arrest its progress. But it is obvious that our remedies are just as powerless against an ordinary catarrh as in cancer; but as a catarrh is not a fatal disease, the surgeon is not specially called on for a remedy. We have become accustomed now to be cured of a bad cold; we must also get accustomed to the natural cause of carcinosis, as also of many other diseases; it need not lessen the pity we naturally feel for these patients, and it ought not to hinder or prejudice the efforts which are being made to increase the knowledge and improve the treatment of the disease. In my opinion much may yet be done in this direction.

The objects which the Surgeon should keep in view are these: the cancerous growth must be removed as early as possible in order to guard against infection, and at least to hinder its progress, and thus lessen the suffering which its presence entails.

If we recognise the cancer disease so shall we strive to find remedies with which to eradicate it. There is scarcely any powerful drug, or variety of diet, or mineral water, which has not been recommended as a sure cure for cancer, and by many believed to really be such. I should have to go through the whole of our materia medica, both old and new, if I were to attempt to tell you all that has been suggested and tried and written on this subject. Like many other incurable diseases, carcinosis has become a camping ground for charlatanism, and even in the latest times there have been those who, by means of some arcana or another, have professed to have a sure remedy for this disease. Unfortunately, this is all and only profession; any truth, which the statements may have contained, was old and well-known before.

The ætiology of cancerous disease, unfortunately, affords no criteria for treatment; we know far too little of the causes which make some tumours infectious and others not so. A blow or contusion may in some cases give rise to the outbreak of the disease, though it cannot produce the predisposition to cancer. In some cases the inheritance of the disease can be proved. Many external circumstances may interfere with the course of the disease, though they cannot produce it. These facts in no way help us in the treatment. There exists at present no specific for carcinosis; but this does not prove that one may not sometimes be found, and still less does it indicate that all internal remedies are useless in the

disease. We must treat such patients whenever any special symptoms present themselves which call for special remedies. Thus anæmia not infrequently accompanies cancerous disease, and, therefore, iron in some one of its preparations will be indicated. In other cases, where nutrition is seriously interfered with, nutrients, such as cod-liver oil and similar things, are indicated; also bitter medicines to strengthen and improve the appetite. Debilitating remedies, diaphoretics, purgatives, mercurials, are to be decidedly avoided, because life will be prolonged in proportion as the patient's strength is maintained and kept up. As regards mineral waters, strong ones, such as Aix-la-Chapelle, Wiesbaden, Karlsbad, Kreuznach, Rehme, are injurious; but milder ones such as Ems, Gastein, Wildbad, may be recommended without fear of causing increased growth of the tumour to take place, provided on other grounds they should seem indicated. Residence in a southerly climate does not seem to benefit cancer patients. Towards the close of life, when the strength begins to decline, a nutritious and easily digestible diet is of great importance, and finally, as the pain increases, a judicious administration of opiates, in some of their various forms, will materially lessen the patient's sufferings. The implication of internal organs will give rise to special symptomatological indications, which I need not here enter upon. Thus much as regards internal treatment, to which I only confine myself, when I am not quite sure of the diagnosis, or in cases which are no longer or not at all adapted for operation.

As regards external treatment, the chief thing, of course, is removal of the tumour, provided its situation renders it possible. The operation may be done either with the knife or with caustics; ligature and écrasement are scarcely ever thought of, excepting, perhaps, the latter in amputation of the penis and of the tongue. Before, however, passing on to consider the advantages of the one or other method, we must settle the question as to the feasibility of any operation, even when this would be easy and free from immediate danger to life, for the views of the most experienced surgeons vary on this point. There are surgeons who never operate in cancer. They say that operations are always useless, because the cancer recurs; if recurrent cancers are operated on the more quickly do further recurrences take place; indeed, surgeons of this school teach that the more frequent the local operation the more quickly do secondary implication of lymph-glands and metastatic cancers

appear, the local tumour, in fact, being a sort of drain of the cancer cachexia; the product of this disease ought not to be removed, because in removing it we favour the outbreak of the disease at other points. If we want to remove the tumour we ought to divert the disease juices to another part, for instance, by the establishment of an artificial ulcer by means of an issue-pea, or of a seton. These views, founded on the old humoral pathology, have not yet been proved to be correct, while experience even shows many of them to be actually false. For our part we can see, by almost daily observation, how development of cancer in the lymphatic glands is brought about entirely by the development of the primary tumour, and we have already, in another place, shown by analogy that the affection of the lymphatic glands in carcinoma is most probably produced by local contagion in some way or other. If after extirpation of mammary or lip cancers, implications of lymphatic glands, which were not appreciable at the time of the operation, become subsequently manifest, we must conclude that the earliest commencement of the lymphatic disease escaped our observation, rather than that it did not exist. How far the existence of a primary and secondary affection of the lymphatics favours or hinders the further course of the disease, the occurrence of metastatic tumours, the general cachexia, is a question which we cannot readily answer, because the course of the disease is not accurately defined; if this were the case, we might compare operated cases with cases where an operation had not been performed, and thus establish something definite as to the results of operations. Approximate results might be obtained from a tabulation of cases which resemble each other as to age, constitution, and nature of the growth; but, unfortunately, the exact differentiation of the different forms of carcinoma and their arrangement is not yet everywhere the same, and hence such a tabulation under existing circumstances would not help us very much; neither would the observation of individual surgeons be sufficiently extensive to allow any reliable conclusions to be drawn from them. In cancers of the face we gain the experience that in spite of the most extensive disease of the lymphatics, metastatic tumours very rarely arise; this strongly suggests that the disease is not potentiated by largely developed local tumour formation, and that carcinoma in the lymph-glands does not increase the disposition to metastatic tumours. The question as to whether carcinoma ever should be operated on or not must be answered in

this wise, that operation has probably no direct effect on the diathesis, and that there must, therefore, be other grounds for operation when we undertake one. We purposely said that operation had no *direct* influence on the course of the disease, because we believe that it may have an *indirect* effect by removing the depression which the tumour itself often gives rise to. The loss of strength, the debility, anæmia, and disturbance of nutrition, which may be brought about by the sloughing and the pain of a cancerous tumour, and perhaps also the constant gnawing anxiety caused by the presence of an incurable disease, are all factors which exercise an unfavorable influence on the course of the disease. I consider it to be the duty of a surgeon, under certain circumstances, to deceive his patients as to the incurability of their disease whenever he considers an operation unadvisable, or when he declines to undertake it. The surgeon, when he cannot remove, ought to relieve the sufferings of his patient, both psychically and physically. Few persons possess that peace of mind, resignation, or strength of character, call it what you will, necessary to enjoy life quietly with the knowledge that they are the subjects of a fatal disease. Patients, although outwardly calm, seldom really thank you for too plain a confirmation of what they secretly suspect. As a surgeon you will often be in difficulties in this respect, and each separate case must be left to your personal good sense, your knowledge of mankind, and your own good feeling. Although we cannot eradicate a disease diathesis by means of an operation,—for instance, although after complete removal of the diseased portion of a mammary gland we cannot prevent the formation of further nodules in another part of the same or in the other, breast (regional recurrence), which up to that time had been quite free from disease, nevertheless, by early removal of the primary tumour, we can prevent the glands from being affected *in continuo*, and sometimes altogether. Even though the complete cure of a cancer of the mammary gland by operation is exceedingly rare, in my opinion, we should get much better results if the general practitioner, to whom these tumours are first made known, would earlier and stringently insist on an operation; as it is, they mostly allow the best time for an operation to pass over, and only send the women to an operating surgeon, when the local spread and the affection of the axillary glands have gone so far that a complete operation is no longer possible. The favorable results which have followed early extirpation of true cancers of the lips and face ought

strongly to induce us to undertake an early removal of cancers in other parts. Although it may have been impossible to operate early and thoroughly, there are, nevertheless, important local causes which may still indicate an operation, in order as long as possible to prevent the spread of cancer to parts, disease of which necessarily entails death. And even although in many of these cases a local recurrence may (will) take place, months or even a year may pass over, during which life is not immediately threatened. Sometimes we have to do with the preservation of parts of the face, as the lips, the nose, the eyelids, which can each be replaced by plastic operation. It is wrong to look upon such operations as useless because they do not cure the disease, for they ameliorate the patient's condition, and make life possible, even agreeable, if only for a short time, and indeed for the whole of the time which he may have to live. We might be very proud if, by means of an operation or otherwise, we could restore an advanced tubercular patient to the enjoyment of life for as long a period as an operation does in many cases of cancerous tumours. In short, there is a series of cases where we can relieve by operation, and I consider it absolutely wrong in many cases to decline to undertake it.

There are other cases, certainly, where it is more difficult to decide. In slowly-growing forms of mammary cancer, as in the connective-tissue cancer, I consider an operation, harmless in itself, as allowable, but not absolutely necessary. But if an eyelid is destroyed, or if the nose, wholly or partially, is lost, then an operation is advisable in the first case in order to protect the globe of the eye; in the second case, in order to obviate a disagreeable defect, the more so, as in these slowly advancing forms of superficial cancer of the face, local recurrence often never takes place at all. Only one circumstance would deter me from an operation—the great debility of advanced age, for then plastic operations to any large extent are not advisable: the unavoidable loss of blood and the consequent illness often suffice to extinguish life. Further, in cases where the situation of the tumour is dangerous, the question of the desirability of an operation may also arise, especially whether an operation is justifiable, which may possibly end fatally, or at least which is just as likely to be fatal as to result in cure. Here the nature of the individual case must decide: we must come to an end with general reflections. The amount of danger of an operation apprehended in any given case will vary

with the experience of the surgeon and the individuality of the patient. This we shall all accept as a principle—only to operate in those cases where, after a careful examination, we can fairly hope to remove all the diseased tissue; a partial operation, leaving behind remains of the tumour, should only be undertaken in very exceptional cases (severe hæmorrhage, very extensive sloughing). There is one point to bear in mind—to always cut well into the healthy structures, at least one or one and a half centimètre beyond the infiltrated part; for only then are we sure to remove all the diseased parts. In a few desperate cases of very extensive cancerous growth life has been prolonged by a desperate operation, but, as a rule, we see more of such cases die than recover.

We now come to a consideration of those caustics which are more especially used in cancerous tumours. Professional opinion concerning caustics has varied very much in the course of years; there were times when a preference was given to them over the knife, and others when they (caustics) were entirely discarded. The views of the majority of surgeons of the present time tend rather to the latter way of thinking, as do my own. For my own part I decidedly prefer a cutting operation, and chiefly on this ground, that I know exactly what I remove, and because I can judge when and if all the disease is completely got rid of. Therefore I look upon the operative extirpation of a cancer and of other tumours as the rule. But where there is a rule so are there exceptions. Thus, caustics may be used in very old persons and in anæmic patients, and if they are well followed up, until indeed all the diseased part is destroyed, the result may be a complete success. From a physiological point of view, at first sight caustics seem to have something in their favour. One would fancy that the caustic fluids would sink into the diseased lymphatic openings, and so destroy thoroughly and surely all local disease. This, however, is not the case, because the tissue, with which the caustic comes in contact, enters into an intimate combination with it, and thus prevents any further flow. It was once thought that recurrence, after the use of caustics, did not take place so quickly as after operations with the knife; but this view has not been confirmed. I can therefore only accept the exceptions above referred to.

As regards the kind of caustic for the destruction of cancers I much prefer to all others chloride of zinc; it may be applied either as a paste or in the form of "arrows." If we have a surface

sore, we can best apply a thin paste, made of equal parts of chloride of zinc and starch, with the addition of a little water. When we wish to destroy deeper parts, we take one part of chloride of zinc and three parts of flour, or powdered gum arabic, and rub them together with just sufficient water to make a thick paste, which we then allow to dry; this we cut with a pair of scissors into little points, the eighth of an inch thick and half an inch long; then we make incisions with a small bistoury into the tumour and insert these little caustic arrows. We repeat the operation until the whole tumour is thickly set with them; they may be placed about two centimètres apart. After this cauterisation, and for about four or five hours, there is moderate—sometimes, however, very severe—pain, which must be combated with hypodermic injections of morphia. On the following day the tumour is found to be converted into a slough, which separates in about four or five days, earlier in soft tumours, and later in harder ones. After separation of the eschar, provided the cauterisation has extended well into the healthy structures, a healthy granulating wound is presented, which shortly cicatrises; if, however, the carcinomatous mass again springs up, a further cauterisation with the paste or arrows has to be proceeded with.

There are these disadvantages to urge against cauterisation—that they are almost always rather painful, and that they are uncertain in regard to the extent of the action. Nevertheless, now and again they are clearly indicated. Vienna paste, arsenical paste, butter of antimony, and chloride of gold, are also well-known caustics. Iodide of potassium, chromic acid, concentrated solutions of chloride of zinc, fuming nitric acid, &c., are less frequently called in use.

I will make a few further remarks on the local treatment of the cancerous tumours, which either are not adapted for or are past treatment. There are cases, unfit for operation, in which the proliferation of the cancerous mass becomes very extensive, in consequence of which the patient is very much troubled and weakened; in such cases partial cauterisations or the actual cautery can well be applied; by means of palliative destruction of the proliferating masses sometimes very satisfactory results may be obtained. The chief indication for treatment in such cases is the horrible and sickening stench which the sloughing gives rise to, and in many cases also the pain. In order to subdue the foul discharges the

actual cautery will be found an effectual remedy; the stench may be lessened by compresses soaked in chlorine water, or acetic acid, creasote, carbolic acid, permanganate of potash, Armenian bole, or dredging with fine charcoal. As your chemistry teaches you, charcoal absorbs various gases readily, and it is on this account an excellent application. Unfortunately it dirties the ulcer so very much that its use has not been very largely adopted. As regards the pain in ulcerating carcinomatous tumours, local narcotics have been tried; for instance, dredging with powdered opium; but subcutaneous injections or the internal administration of narcotics is found to be more certain, and so at last we generally order morphia for these unfortunate patients. Perseverance in the treatment of such cases and in relieving their sufferings is a duty I strongly insist on; it certainly is sad for a surgeon to be able to do so little, but that little we ought never to grudge to these hopeless patients.

Some short remarks on the Clinical Diagnosis of Tumours.

I cannot wonder if all that I have told you about tumours is at present somewhat perplexing to you; it may possibly console you to know that I was not less perplexed when I was in your present position. Nothing but a longer study of the subject and practice in diagnosing the various varieties of tumours, which this clinic affords, has made it possible to speak with any degree of certainty on this difficult subject. The consistence of tumours, their appearance, their relation to the surrounding parts, their localisation, their rapid or slow growth, the age of the patient, are all points through which to form an opinion; and first one and then another of the above-named conditions decides the issue. Let us take a special case as an example: a man, about fifty years of age, presents himself before you, strong, and ruddy for his age; for many years he has had a tumour on his back, which formerly did not cause him any inconvenience; only since it has attained the size of a child's head has it become inconvenient. The tumour is soft and elastic, yet not tense, nor fluctuating; it is movable beneath the skin; the skin is unaltered in appearance; the tumour has never been painful, and it is not tender on being examined. The diagnosis in this case is very easy; the tumour is almost certainly a lipoma, partly on account of its position, partly because it is situated in the subcutaneous

connective tissue, and partly on account of its slow and painless growth. It might possibly be a soft connective tissue tumour, but in every probability it is a fatty tumour. Let us instance another case: a woman presents herself to you with a tumour in the breast; this tumour is hard, knotty, and about the size of an apple; on its surface there are small spots of retracted skin, and the skin is adherent to the tumour. From time to time there have been darting pains in it; on pressure it is tender; the axillary glands of the side corresponding to the mammary tumour feel hard. The woman is about forty-five years of age, well nourished, and appears healthy. In this case, also, the diagnosis is easy. We have no doubt to do with a carcinoma—(1) because at the present age of our patient cancerous tumours of the breast develop most frequently, whereas adenoma and sarcoma usually occur much earlier; (2) their consistence might have led to the diagnosis of fibroma, but the fibromata are exceedingly rare in the mammary gland; (3) carcinomata are painful, as was this case, while sarcoma and fibroma are usually not so. We might adduce other points in support of our diagnosis, but what has been said already suffices. Let us just take a third case: suppose a child, ten years of age, has a slowly-growing, rather painful swelling of the middle third of the lower jaw, which has been coming for the past two years; the teeth on this side have fallen out without being diseased; the swelling of the bone is uniformly round, and extends from the first molar of one side to the corresponding point of the other; below it is bony hard, above (inside the mouth) it is covered with mucous membrane, and elastic. Can such an osseous swelling be due to a chronic inflammatory process or to necrosis? This is hardly probable—(1) because the pain has always been slight; (2) because there is no suppuration, which is hardly likely to fail after an ostitis of the lower jaw, which has gone on for two years; (3) because the swelling is so localised and so uniform, neither of which things occurs in caries or necrosis; (4) and because ostitis of the lower jaw does not often occur in patients at this age, except after phosphorous poisoning, which was never a question in the present case. Thus we have to do with a tumour. Is it an osteoma? Is it too soft on its buccal surface, for it can easily be pierced with a fine needle? Is it an enchondroma? Consistency, form, mode of growth, age of the patient, all fit in well, except its position. Chondromata of the middle third of the lower jaw are very un-

common at this age. It is a central osteo sarcoma, probably a giant-cell sarcoma; all the symptoms fit in exactly, and, besides this, you know that these tumours are frequent in the lower jaw at this period of life. I say that "you know;" it would be better to say that you will come to know it by degrees, and I can only advise you, after every time you see a patient in the wards with a tumour, to go home and read up the case, and to compare the individual case with the general characteristics of tumours which I have pointed out to you. After having done this for some little time, and after having examined under the eyes of your teachers into the pathological histology of all the tumours you meet with, you will soon get a clear view of the subject, and the various peculiarities of each will then become fixed in your memories.

LECTURE L.

CHAPTER XXII.

ON AMPUTATIONS, EXARTICULATIONS AND RESECTIONS.

Importance and signification of these operations. Amputations and exarticulations; indications; methods; after-treatment; prognosis. Conical stumps; prothesis; historical. Resections of joints; historical; indications; methods; after-treatment; prognosis.

Gentlemen,—We have frequently had the opportunity of speaking of amputations and resections, and before bringing these lectures to a close I should like to give you some idea of these very important operations, by means of which we remove diseased limbs or portions of limbs, the healing of which we are unable to bring about. These successful, and in many cases life-preserving operations, have been considered rather as a "*testimonium paupertatis*" of medical art, for the cutting off of diseased structures is not really a healing, that is, if by the art of healing we are to understand the art of restoring a diseased tissue to its normal condition. Yet, if you try to apply this undoubtedly very high standard to all cases, the confines of what is curable are reduced to a minimum. Thus you might say a cataract is not curable, for the opaque lens is not rendered transparent—it is removed; and further, you will have to regard some of the most brilliant cures which dermatologists effect by means of external caustics as so many proofs of the powerlessness of our art; also the preservation of a man's life by the removal of a tumour from the larynx will come within the same category. The most brilliant cures, in the strictest sense of the word "healings," are made in syphilis, for instance, with anti-syphilitic internal treatment. We can bring about, as if by magic, the absorption of wide-spreading diseased products in the course of a few weeks which have

lasted for a considerable time. Such remarkable cures are, however, very rare in other diseases, and we are often obliged to content ourselves by destroying the diseased parts, in order not only to prevent the spread of disease to the surrounding parts but also to guard against its affecting the constitution at large.

The less important for the life of an organism a diseased part is, the more readily do we decide to sacrifice it. On the other hand, the greater the part that has to be removed the greater also will be the danger connected with its removal, and the greater will be the loss of the endurance power of the individual affected. This condition introduces among the indications for amputations a non-scientific social element, which not infrequently helps us to a decision. Thus, for a rich man it would be possible to live, and to a certain extent to enjoy life, even if he were to lose all the extremities, for that which the extremities have physiologically to contribute to the existence of an individual can be supplied by the work of other individuals, and work can be bought. But for some one, whose living depends on his hands and feet, the loss of an extremity, or even the crippling of a single finger, becomes the possible source of destruction of his future social existence. Thus, for instance, what is a letter-carrier, mason, or carpenter without sound legs? What is a goldsmith, watchmaker, or shoemaker with one hand gone? I have often been called on to remove a sound finger that has become contracted on to the palm of the hand, because it prevented the man from holding his hatchet or his spade, which his trade required of him. How many times I have heard used the reproachful words, "Oh! so you cannot cure my leg! well, then, I prefer to die than have it removed, for what could I do without a leg? I should be a ruined man! No, I will never consent to that."

Nevertheless, dying of chronic diseases of the extremities is sometimes exceedingly slow; but the daily suffering going on for weeks, or months, maybe for years, apparently without any end, humbles the strongest man; then the desire to live, becoming gradually reconciled to the idea, the possibility of being able to fill some employment even after the loss of a limb, generally induces patients to submit at last, when it is sometimes too late, however, to amputation. The amount of opposition which seriously injured persons display towards amputation varies much. In one case the appearance of the injured member and the amount of pain help to decide. In another case, when an extremity is torn into shreds,

and when the unfortunate man himself sees the broken fragments of bone lying about, we shall meet with little opposition to amputation; the same may be said of cases in which there is very great pain, with loss of power over the fingers or toes. But the case is far different when the extent of the injury is visible only to the surgeon; for instance, where there is an injury to a joint with fracture or considerable dislocation of a bone, but without primary disturbance of function (as when the patient can move his toes and fingers, although they may even be considerably damaged), and when there is not much pain. In such cases it is sometimes very difficult to convince a patient of the necessity for operation; he must have unlimited confidence in his surgeon; we may even say the patient must believe in the surgeon as something more than a human being if he allow the necessary primary amputation under such circumstances.

You will often experience how all the principles of your surgical treatment, learnt from long and extensive practice, are at first absolutely set at defiance. Then after a few days, when the consequences of the injury begin to assume the dangerous character which you prognosticated, and when the patient himself implores an operation, you will occasionally be obliged to reply, too late! Will you venture to recall to the man, under such circumstances, his previous obstinacy? These are hard times for the surgeons. But if there should be ever so slight a chance of saving him, we must not hesitate, even now and under unfavorable circumstances, to undertake amputation. The hope of saving a life, which is despaired of, even in spite of such a combination of hopeless circumstances, is a glorious proof of the strength and power of our art. But when one unsuccess follows another, we grow weary of striving, under such unfavorable circumstances after that which is so seldom attainable; for however gratifying it is to accomplish extraordinary deeds by extraordinary powers, we must be careful not to bring the means which our art possesses too often into the danger of being fruitless before our own eyes or the eyes of suffering mankind. For too many failures finally stamp out from the mind of every conscientious surgeon that pleasure and that confidence in his own science which is absolutely necessary for success.

I hope that what has been said will suffice to induce you to consider earnestly before each great operation, and especially before each amputation, whether you shall operate or not. Picture to

yourselves, that in each capital operation you demand from your patient that he place his life absolutely in your hands, and that you are responsible to him for it; then let him have the fullest benefit of your best powers.

It is rather difficult to state the general indications for amputatations and resections. Almost each general statement might be criticised in any special case. Nevertheless, it is convenient to have a systematic doctrine, and hence I shall briefly formulate the teachings which I have inculcated in the course of these lectures. I will then add a little concerning the principles which ought to guide us in the technical performance of these operations, as well as in the after-treatment which the patients require.

Amputations and Exarticulations.

There are injuries of the extremities, in consequence of which it is clear from the very commencement that the extremity will become grangrenous; or that the consecutive suppuration will be so extensive and so unfavorable that the life of the patient will necessarily be placed in great danger. If, however, we should be prevented by the obstinacy of the patient from carrying out primary amputation, an operation performed after propressive gangrene had set in, or in case of an extensive phlegmon with septicæmia, would scarcely be able to avert death. It is only where we can amputate in healthy structures that there is any prospect of success; as for instance, in traumatic gangrene, which has spread from the hand or forearm as high up as the elbow-joint, we must amputate high up in the arm or exarticulate at the shoulder-joint, if we would be successful. The results of a high amputation through the thigh under analogous circumstances is far less successful, because this operation in itself is far more dangerous than corresponding ablations in the upper extremity.

If conservative treatment have been successfully pursued for some time, and then symptoms of pyæmia show themselves, amputation may be resorted to, and in the upper extremity it may be successful; under similar circumstances, it would be less successful in the lower extremity. A more successful result will probably be obtained from so-called secondary amputations, provided there are no symptoms of pyæmia; in cases where, in consequence of extensive phlegmon, large areas of skin have sloughed, so that it is impossible to close the wounds, or where, in consequence of chronic

suppuration of large joints, a condition of general marasmus may set it.

Injuries of the hands or feet may give rise to primary amputations, when they are such as, even after the most favorable conditions of healing, it is probable that an useless or irritable stump will result. Especial, after avulsions and severe contusions, the wounds may be of such a kind that the bones project; then the wounds must be refinished *lege artis*. The same rules will hold good after frost-bites. But when it concerns the lower limb, amputation must not be postponed too long, if the line of demarcation is clear. The sloughing off of large portions of the body is very often accompanied by sepsis, which an early amputation, in cases of gangrene after frost-bites or burns, may possibly obviate. As regards the acute, non-traumatic inflammation of bones or of joints, we are constantly being taught how to save the limb by early diagnosis and treating the disease by favorably placed counter-openings for the escape of the pus, by fixation, and by suitable positions of the limb; on the other hand, however, there are cases where the patient can only be saved by an early and timely amputation. Undoubtedly the choice of the right moment for operation is very difficult, seeing that it involves a knowledge of how long, and whether the patient can bear the suppuration and the hectic condition to which he is reduced.

Concerning so-called spontaneous gangrene, or, as it was called by the older surgeons, gangrene from internal causes, we must distinguish each individual case. If the gangrene has resulted from arterial embolism, we must amputate—provided the general condition permits—as soon as the line of demarcation of the gangrene is perceptible. In gangrene after typhoid and severe attacks of the exanthemata, we may wait until the patient has somewhat recovered his strength. In pure gangrena senilis amputation is seldom indicated. If the gangrene confine itself to one or several toes, we wait for spontaneous amputation; if it (the gangrene) spread to the foot, it seldom afterwards limits itself; but if limitation should take place, then we ought to remove the projecting bone with as little injury to soft parts as possible, in order to secure enough to make the necessary flaps to cover the stump.

Of chronic inflammations, it is before all, those which attack bones and joints which lead to and require amputation. Long standing caries of several bones of the wrist or ankle, caries of the knee-joint in non-tubercular adults, caries of the hip-, shoulder-,

and elbow-joints, rather require resection—if any operative treatment is required at all. Amputation in such cases only ranks in the second line.

Extensive incurable ulcers of the foot, and incurable ulcers of the shins—or ulcers which break out again almost before their cure is complete—often require amputation, unless such individuals are to be condemned to perpetual pain and suffering, or confined permanently to their beds.

Large aneurisms of the femoral artery which are about to burst, and which cannot by any other means be cured, would certainly lead to death unless amputation were to be timely performed.

Amputations must also be performed in tumours of the extremities which are grown firmly to the femur, or humerus, or tibia, and have infiltrated into and between the soft parts. Tumours which are attached to the ulna, or radius, or fibula, and have not grown very deeply into and among the soft parts, can be successfully removed either by partial resections, or even by local extirpation of the affected bone.

Finally, deformities and contractions of the feet may call for amputation if the individuals are unable to walk in consequence of them.

As regards the manner of performing amputations, they may either be done at and through a joint, or the bone must be sawn through. Each method has its advantages and its disadvantages. The removal at a joint seems almost to be the natural method, being less serious and simpler. The soft parts may even heal *per primam intentionem* over the cartilage, or the latter may necrose and suppurate, healing taking place by means of granulations which spring from the bone. In this way the medullary cavity of the bones is not opened, and a possible primary infection of the medulla is thus entirely or almost avoided.

The remains of portions of synovial membrane are an unfavorable circumstance, because they show no tendency to primary union, and even after the wound has closed they are always liable to become the seats of collections of pus; further, the soft parts which are intended to cover the large joint surfaces must be very ample, and thus the wounds are very extensive. At the elbow- and knee-joints, with the same length of flaps, we might perform amputation high up in the forearm or in the leg. From a technical point of view, the stumps after exarticulations are less favorable to the

adaptation of artificial limbs; because the joint of the artificial limb comes to be on a lower level than the healthy joint, as, for instance, at the knee. There is this advantage in amputations, that we can more easily choose the point at which to remove the limb, if either on empirical or prognostic, or prothetic grounds we incline more favorably to this or that spot. As a general rule, we require a less amount of flap to cover-in an amputation than an excision. Sawing through the bone after all is not such a very serious complication of this operation, although in many cases a slight necrosis of the sawn surface does take place. If the medulla, either in the medullary cavity or in the spongy substance, gets infected at the operation, either by impure sponges, or from retention of pus in consequence of becoming adherent to the soft parts, then a more or less severe osteo-myelitis may set in, which may even lead to death through septicæmia. In more favorable cases, the osteo-myelitis is limited to a certain height, and then extensive necrosis of the shaft of the bone takes place, and after six or eight weeks the sequestra may be removed from within a sheath of newly formed bone which then takes its place. We have already pointed out, when speaking of compound fractures, how osteophytes form at the extremity of the bone, and close in the medullary cavity. Osteo-myelitis of an amputation stump is at first very difficult to recognise. You may pretty certainly suspect it if about the third day after the operation your patient (till then quite free) gets very feverish rather suddenly, and is then attacked with rigors and diarrhœa, although the stump shows no symptoms of inflammation, having indeed healed almost by the first intention. The cause of the fever, thus not being due to inflammation of the soft parts, must lie in the bone if other complications are absent. At all events, under such circumstances, you must open up the wound, lay bare the medulla, so that any pent-up pus may easily escape. You will sometimes save the patient thereby, but it is generally too late, because, on account of the obscurity of the symptoms, one seldom has the courage to lay open the beautifully healed wound in the soft parts, although strictly speaking the state of things is not rendered worse thereby, even if the diagnosis was incorrect.

In the performance of amputations and exarticulations it is above all things important—

(1) To perform the operation with as little loss of blood as possible;

(2) To arrest the hæmorrhage so completely that there shall be no fear of secondary hæmorrhages; and

(3) To cover the bony stump so thoroughly with soft parts that the latter may easily and completely heal and unite over it.

As regards the first two of these points, I have nothing to add to what I said formerly about them. Before the operation, the vessels must be emptied of their blood on Esmarch's plan. The amputation may then be performed without the loss of a drop of blood. After the operation, I employ torsion for the small visible arteries and close the larger ones by means of acupressure or a catgut ligature; after the exarticulation of the femur or humerus, I tie the femoral or axillary artery because, in my attempts to apply acupressure there also, I have not succeeded quickly enough in fixing the needle as firmly as it is necessary to do.

The bony stump must be covered with soft parts, and these must heal and unite over or in front of it; if this does not take place and the bone projects, then the granulations growing from it either do not cicatrise at all, but become converted into an ulcer, or even if cicatrisation takes place, the cicatrix attached to the bone has so little power of resistance that it rapidly breaks open again, and continues so on the fitting to it of a wooden leg or artificial foot. The patient is then in bad plight, and must give up all hope of using the stump and go about on two crutches for the remainder of his life suffering pain from the ulcers.

The bone must, therefore, always be sawn off above the point at which the incision was made through the soft parts. In cases of exarticulation, the soft parts must be cut through deeper than the end of the bone which is left. On this principle we may cut through the soft parts in the following manner, and give them the proper form for covering the stump.

1. We make the *circular incision, i.e.* we cut in a circle round the entire limb, draw the soft parts which have been cut through forcibly back, and then saw through the bone; the soft parts are then set free and fall together over the bony stump. To attain the object in view suitably and certainly in this manner, the following is the best mode of proceeding: we first cut the skin all round completely through, then dissect it off in such a manner that as much as possible of the subcutaneous cellular tissue and panniculus adiposus shall remain in connection with it, but leave the fasciæ of the muscles upon them. When this dissection has been carried

out all round to the extent of 0·8—1·6 inch, the detached skin is turned back and drawn strongly upwards with the rest of the soft parts by an assistant; we now make another circular incision through the muscles down to the bone at the point to which the skin has been turned back; the assistant who holds the stump now places both his hands upon the divided muscles and draws them as far upwards as possible; with a third circular incision we now cut through the deep layers of muscle about 0·8 inch above the level of the first incision through the muscles, again down to the bone, then detach the periosteum and saw through the bone at this point. When the bone has been sawn through and the drawn up soft parts allowed to fall into their natural position, three incision-strata must present themselves, namely, that of the skin, that of the muscles, and that of the bone, which latter lies at the bottom of the funnel-shaped wound. In emaciated limbs the soft parts should project about 2·4 inches beyond the stump, in muscular ones about 3·2—3·6 inches. If a forearm or leg is to be amputated, the muscles between the bones also must be divided carefully, after the last cut round the bones has been made, before the bone is sawn through.

I consider it advisable for you to practise the circular incision first of all exactly as I have just described it to you, and to accustom yourselves to make very smooth incisions and, above all, to learn to bring about the action of the knife, not by pressure, but by drawing it forwards. I by no means wish to assert, however, that the circular incision cannot be made suitably in any other manner. The following modifications are admissible under certain circumstances; they refer partly to differences in respect to the eventual form to be given to the stump, partly to differences in the technical procedure for the attainment of the result described formerly.

We may amputate an extremity in one plane, as if with an axe or guillotine (Botalli); this may be carried out successfully for the fingers. We generally prefer exarticulation to amputation in the case of the fingers, but cases occur in which fingers are cut straight off by machinery (circular saws, straw-cutting machines), and it then becomes a question whether such a stump is already well formed without further surgical interference. This is, in fact, the case, but it is dependent upon the peculiar anatomical conditions in the fingers that the skin which is attached to the sheaths of the

tendons and to the bones scarcely retracts at all, while the tendons themselves are drawn back into their sheaths. At most of the other points of the extremities, not only is the skin so movable over the fasciæ, but also the muscles upon the bones, that after a simple transverse amputation in the same plane not only the muscles about the bone, but also the skin would become strongly retracted. Since this *circular incision in one plane* always leads to conical amputation-stumps except in the case of the fingers and toes, it is scarcely ever performed now.

A similar limited use is made of the *circular incision in two planes*. A flap of skin is formed, and then the muscles and bones are divided in one plane. In this manner the bony stump is covered with skin only. Where many muscles surround the bone, they will retract strongly and take the skin back with them with this method, and the end of the bony stump will come to lie in about the same plane with the cut skin; during the healing process the skin then grows so closely to the conical surface of the divided muscles that the bone projects and a conical stump is again formed. This method is admissible at parts of the extremities only at which the muscles do not retract around the bone, because they and their fasciæ are attached to each other and to the bone either abnormally or in consequence of previous disease of long standing, *e.g.* in amputation of the leg close above the malleoli and immediately under the head of the fibula, or at the analogous points of the forearm, but the flap of skin must then be made long enough to cover the stump well.

The *circular incision in three planes*, that first described, by which the skin, muscles, and bone are divided at three times, in these different planes, may be carried out in various ways. For the commencement of your practisings on the dead body I would recommend you to proceed as I pointed out at first. Instead of the last incision through the deep layers of the muscles, you may push back with a rasp the periosteum in the plane of the first incision through the muscles, upwards to the extent of 0·8 inch, and then saw through the bone, the effect with regard to the formation of the stump remaining the same. Whether the deepest portions of the funnel are lined with periosteum or with soft parts about the bone (parostal) has no influence upon the healing and formation of the stump. With a little practice you will soon learn to divide the layers of muscle in such a manner as to give to the funnel the

depth and form you wish. If, however, your assistant draws the soft parts too strongly back, and you cut through thin layers only of muscle always higher and higher up, you eventually come to saw through the bone much too high up and will have by far too large a quantity of soft parts in front of the bony stump; if your assistant applies too little force, or if the soft parts, on account of their adhesion to each other or to the bone, cannot be drawn back properly, while you cut through the muscles too rapidly and too deeply, you will have a deficiency of soft parts and eventually a conical stump.

The circular incision is the normal method for all amputations, it is applicable to all points of the extremities, although flap and oval operations are often more practical for exarticulations.

2. *Flap operations.*—We form of the soft parts one or two flaps with which to cover the sawn surface of bone. If we form one flap, whose base usually comprises half the circumference of the limb at the point of amputation, a circular incision in one or two planes is generally made in the other half. In the case of flap operations also, it is advisable before sawing through the bone to push back the periosteum 0·4 inch, and to saw through the bone 0·8 inch above the base of the flap, so that, when the muscles retract, the bony stump shall not press too much against the inner surface of the flap laid over it.

I prefer forming the flaps so that, when the extremity is placed in position in bed, they will hang over the wound without any support by means of sutures. The flap should consist of skin at its lower, of skin and muscles at its upper part. The most practical way of effecting this is first to produce the form of the flap by means of incisions through the skin which extend as far as the fasciæ beneath; the flap of skin is then to be turned back somewhat and at its new boundary the muscles are to be divided down to the bone in a similar form. The circular incision in two planes is then to be made at the posterior part of the limb. The length of the flap should be about one third of the circumference of the limb at the point of amputation; the base should comprise one half of the circumference, rather more than less.

The single flaps have the advantage that, in the case of irregularly formed wounds from injuries and irregular forms of edges of ulcers and lines of demarcation in gangrene, we can sometimes amputate lower down than if we performed the circular incision,

whereby not only may a longer stump be formed but the prognosis will also generally be more favorable.

The *formation of two flaps* has, in my opinion, no advantage over the circular incision. If we form two lateral or one upper and one lower flap, the quantity of soft parts and their form is always analogous to those with the circular incision. It sometimes happens with the circular incision that infiltrated skin can neither be easily drawn back nor formed into a flap and turned back; we then divide the skin above or below in the long axis of the limb; flaps then result from the circular incision which assume the funnel shape.

It is not advisable to form flaps *of skin alone* with which to cover the stump, for long flaps of that kind readily become gangrenous at their edges, and if there is no muscular tissue between the skin and the sawn edge of the bone, the latter often causes ulceration from within outwards, which perforates the flap. This is not indeed, in itself, any great misfortune, for the sawn edge of bone exposed either becomes necrotic and detaches itself, or it soon granulates and becomes cicatrised. In both cases, however, adhesion takes place between the cicatrix and the bone, and gives rise, later on, when the stump comes into use, to troublsome ulcerations.

The method of forming the flaps by thrusting in a long pointed knife, as if we wished to pierce the limb in the middle from above downwards or from one side to the other, and then passing the knife over the bone out at the other side and pushing it gradually out, generally results with beginners in the formation of a very muscular and sometimes too pointed flap, which is deficient in skin and cannot be adjusted conveniently over the wound. With experience and practice, however, good flaps may be formed in this way also.

The flap operation is applicable at all points of the extremities, but not practical everywhere. By the aid of drainage tubes we may draw off the secretions well, also with flaps formed from below upwards. If the flaps do not heal after the amputation to a great extent by the first intention, the after-treatment is always somewhat tedious, because we have to prevent them from becoming rolled inwards by cicatricial contraction.

3. There is a third method of amputation still in use, by means of which the wound assumes a form between that with the circular and flap operations, namely, the *oval operation*. The incision-plane of the oval lies obliquely from above downwards; the upper part

of the oval receives a more pointed form, the lower is more rounded. After the incision has been made in the skin, the latter must be drawn forcibly back, and the soft parts and bone divided on the same principles as in the circular operation. For amputations, the oval operation has gone almost entirely out of use since it has no advantages over the circular and flap operations. For exarticulations of the fingers and toes in the metacarpal and metatarso-phalangeal joints, of the pollex pedis with the first metatarsal bone, and of the thumb with the first metacarpal bone, the oval operation is very useful. For exarticulations in the shoulder- and hip-joints I should perform it only when there is not sufficient skin for the formation of flaps.

As regards the *preparatory arrangements*, *assistance*, *choice of instruments*, and *after-treatment* in operations, I have still something to add.

While the patient is being narcotised, or before—for many individuals are narcotised with difficulty if their attention is kept up during the process of narcotisation by manipulations of the diseased part about to be operated on—we clean the extremity carefully with soap and water, especially in the neighbourhood of the intended operation. The bandaging for the prevention of hæmorrhage is then carried out and the bandages removed again with the exception of the upper ring. One assistant now takes hold of the upper part of the limb, another of the lower part. In cases of amputation, the operator stands so that he can eventually push up the soft parts with his left hand in common with the assistant, and that the amputated limb shall be separated to the right of him; in cases of exarticulation, the operator must stand so that he can himself control with his left hand the movements of the limb to be exarticulated.

For amputations and exarticulations of the toes we use small knives from 1·6 to 2 inches long. For exarticulations of the hand and foot, as well as for amputations in the lower half of the forearm and leg, we use knives with a blade 6 inches long; for the upper part of the forearm, the arm, the upper part of the leg, and the lower part of the thigh, we use knives with blades from 6 to 10 inches long; for the high amputation and exarticulation of the thigh, we choose knives from 10 to 14 inches long. If you have for your practice two small knives with blades 2 inches long, and one with a blade 6, 10, and 14 inches long respectively, that will

suffice. I am not fond of changing knives during an amputation, and therefore like to have the point of the cutting edge rounded off a little, so that I can dissect off the flap at the commencement with it; other operators prefer small rounded-off knives for this purpose, such as are used for the incision in tying vessels, then others for cutting through the muscles, and still others for detaching the periosteum. I use a broad rasp for pushing back the periosteum, and this may sometimes be effected with the nails alone. A skilful assistant will be able to draw back the soft parts with his two hands sufficiently for the operator to carry out the necessary cutting and sawing without endangering that assistant's fingers, but we may also employ large pieces of clean linen for that purpose. Many operators take pleasure in performing amputations of even very fleshy limbs elegantly and quickly with the smallest possible ordinary knives, and thus pushing the simplicity of the apparatus of instruments to its utmost limit. All these things are, if not altogether unimportant, very much dependent upon custom and tradition, and every one may follow his own taste therein. After sawing through the bone I generally take off the sharp edges of the sawn surface with a bone foreceps.

When the amputation has been completed, the vessels must be secured by torsion, closed by acupressure, or tied. All the instruments and ligatures necessary for that purpose must be near at hand. First of all we close in one of the ways just mentioned the recognisable arterial openings; the elastic ring must then be loosened somewhat, but in such a manner that it can be secured again immediately if much hæmorrhage occurs. Arteries seen bleeding after the removal of the elastic ring or tourniquet should be closed by acupressure or tied. Venous hæmorrhages occur in amputations of the arm or thigh since the valves there seldom suffice to prevent them. These veins should be tied or closed by acupressure. I look upon torsion of the veins as dangerous. Very troublesome are arterial hæmorrhages from the medullary canal of the bones; they are seldom profuse, but any disturbance of the medulla with forceps, or a firm pressure with pieces of sponge is hazardous; the employment of styptics, especially of Liq. Ferri, should be avoided altogether. I should advise you not to take any notice at first of this hæmorrhage, which usually ceases of itself, until all the other arteries have been tied; should the hæmorrhage not cease, the main arterial branch

of the limb may be compressed for a time with the finger, which will cause a gradual stoppage of it. For cleansing the wound during the tying of the arteries, only entirely new, soft sponges should be used.

After waiting until the hæmorrhage has ceased entirely, it is quite proper to leave the fresh wound exposed for a time to the air. Wounds caused by the circular and oval operations are generally closed vertically. I always introduce 2 to 4 sutures only into the upper part of the wound and leave the lower part open. Flaps I fix by means of 2 to 4 sutures in the position in which they are to adhere, and place previously a drainage tube dipped into glycerine in the wound, obliquely in front of the bone, so the two ends of the tube may project from the corners of the wound. I apply no dressing.

The stump is placed in the bed in such a manner that the secretions from the wound may flow into a vessel placed beneath it without wetting the bed. In 2 days the acupressure needles may be removed; in 6 to 8 days the drainage tube may also be taken out. In cases running a thoroughly normal course, the stump should never swell nor the patient have any fever. In 10 to 14 days the parts of the stump not yet healed may be covered with disinfected lint and the stump enclosed in a cloth dressing only, so that the patient may move somewhat more freely in bed.

If the stump does swell, however, or if the patient has violent fever, the adhesions in the wound must be separated with the finger, and any cavities in the wound in which pus has collected and become decomposed laid open. If violent *neuralgic pains* set in, with frequent *twitchings* in the stump, subcutaneous injections of morphia must be used.

If *secondary arterial hæmorrhages* occur within the first twenty-four hours, the artery must be looked for and secured. If such hæmorrhages occur later (in the second or third week), with a granulating wound, it is then also best to attempt, first of all, to discover the bleeding artery and secure it. If this cannot be done and the hæmorrhage returns after long-continued digital pressure, the main artery of the stump must be tied.

Many surgeons prefer closing the wound accurately immediately after the operation and putting on a dressing which keeps the soft parts firmly in contact with the bony stump. Others fill the entire cavity of the wound with lint, which is dipped eventually into

styptics, and bring the soft parts together over it with a dressing which is not to be removed for forty-eight hours. I have not seen any good results from either of these methods; neither the attempt to force on union by the first intention nor to cause from the first intense suppuration is advisable. With the open treatment of wounds complete union by the first intention may take place; if suppuration takes place at most points of the wound, the pus may escape easily unless too strong adhesions have occurred early. The surgeon must learn from observation how best to effect this.

In Lister's method the wound is thoroughly cleansed, but drainage tubes are always introduced, as I have mentioned already, and then a light dressing applied with slight pressure, which is renewed at first, however, as often as it becomes saturated with blood and serum. Quite recently I also have treated some amputation-stumps on Lister's plan and obtained good results, and the majority of German surgeons now employ this method almost exclusively.

The treatment of amputation-stumps in the water-bath encounters so many technical difficulties that it was soon given up again.

We have already, when speaking of the circular amputation in one plane, alluded to the very obnoxious *conical amputation-stumps*. They may depend upon injudicious incisions through the soft parts, or upon a deficiency of soft parts for covering the stump. This is not, however, either the only or yet the most frequent cause of their formation, for there sometimes occurs in atrophic individuals such atrophy of the soft parts of the stump that they continue to become thinner and shorter, and more and more retracted from the bone; the latter occurs especially in the lower portion of the femur, where but few muscles are inserted and none have their origin. Inflammations and suppurations of the stump also, even with ample soft parts, result therein that the intermuscular and parostal inflammatory infiltration leads to a process of contraction which draws back the soft parts and attaches them to the bone so forcibly that the cicatricial contraction of the granulations of the wound is insufficient to overcome it. This is the most frequent cause of the formation of conical stumps. Since processes of inflammation cannot be prevented, the surgeon cannot by any means always be held responsible for the formation of bad stumps. It might be thought that this imperfection would easily be avoided by retaining an ample supply of soft parts for covering the stump, but an excess of soft parts in a fresh stump has also considerable disadvantages.

If very long flaps have been formed consisting of skin only, these will become gangrenous at their extremities. It is then not the loss of soft parts which is most prejudicial but the process of decomposition in the fresh wound. This must also be prevented if possible. If too long muscular funnels or flaps have been formed, another disadvantage presents itself, namely, that the soft parts are then so heavy that they hang down greatly about the stump and become pressed by their weight against the edges of the sawn surface of the bone. In such cases we may place the wound under more favorable circumstances by placing a splint beneath the stump and thus giving support to the dependent soft parts.

If we see that a conical stump is forming, we may attempt by means of adhesive plaster and traction with a weight, as in coxitis, to draw the skin gradually forwards, or at least to assist the concentric contraction of the granulating surface by freeing it from the counter-traction. If the patient can bear this without suffering pain about the stump or becoming feverish, it may be of use; if these symptoms show themselves, we must abandon this method. If extensive necrosis of the stump should set in as a consequence of previous osteomyelitis, the stump will thereby be rendered somewhat shorter, it is true, but the osteophytes which had formed around the bone prevent it from collapsing, and themselves become atrophied very slowly in the course of years; my experience does not tend to show that the conical form of a stump is obviated by the throwing off of a sequestrum. Some kind of operative interference is generally necessary. I lay open the cone of granulations upwards into the skin or thereabouts, downwards as far as the bone, then push the rasp along the bone and separate the periosteum together with the osteophytes so far inwards into the soft parts that these may cover conveniently the bony stump to be formed further back. I use a chain saw for removing the bone, the ends of which I hold above the bone while the cutting part surrounds the bone from below. In limbs with two bones, this subperiosteal resection or amputation is carried out in a similar manner for both bones. Care must be taken that the secretion from the periosteal canal from which the resected piece of bone has been removed can escape freely; this canal has a great tendency to close in front by the first intention, in which case pus may collect in it and become decomposed, giving rise to ichorous osteomyelitis. I was unfortunate enough to have such a case in the military hospital at Mannheim in

the person of a soldier who had got well over a dangerous injury of the knee and amputation, and eventually succumbed to my great regret in the manner just mentioned, since this danger of subperiosteal resection in amputation-stumps was not then known to me because all the cases previously treated by me in that manner had run a favorable course.

Observation of amputation-stumps has shown that they do not undergo much change in the course of time. Many stumps become enormously emaciated; very muscular flaps become so greatly atrophied from inaction that nothing but skin remains. The majority of stumps become conical in the course of years, even though they are covered with skin; this occurs most certainly in weakly and atrophied individuals, and especially in those in whom amputation has been performed for caries of joints and who subsequently become affected with caries in other bones or in the stump itself, or with pulmonary tuberculosis or lardaceous disease. The bones of such stumps become atrophied and their cortical layer thin. Short stumps of the thigh form almost the only exception to this. If these are much used for walking, the muscles which pass from the pelvis to the thigh become strongly developed, the skin also participates in this good nutrition, and the stumps become stronger than they were immediately after the operation. From the circumstance that the generality of old amputation-stumps are covered with skin only and that the muscles have disappeared, the conclusion has been drawn that it is quite unnecessary to employ muscles for covering the stump. That this is not favorable for the healing process has been mentioned already.

Neuromata of amputation-stumps have been spoken of already.

As regards the *prognosis for amputations*, all that can be said thereof in a general way is that they are the more dangerous the more closely they are performed to the trunk of the body, but a great deal also depends upon the general condition of the patient at the time of the operation. Amputations on account of injuries are always followed by less favorable results than those on account of chronic diseases, but so many circumstances concur in each individual case that we have not time here for the discussion of points the general importance of which is only very conditional.

Surgeons pay much too little attention on the whole to the after fate of amputations and to the prothesis. In your practice you will hear many complaints from such patients. The most frequent

of these are of pains in the stumps on every change in the weather of excoriations in the cicatrices, of pressure of the wooden legs or other artificial appliances at one point or another, and of constant repairs required for the latter. Many also suffer very much from the feeling, which sometimes endures for years, that the limb removed is still present in its entirety. They assert, for instance, positively, after amputation of the thigh, that they have the sensation of tearing in the great toe, or that the foot is in a bad position, &c. In the first days and weeks after the operation, these sensations are the rule, and are so distinct and strong that by covering the stump we might easily deceive the patient for weeks concerning the loss of his leg, and I have even seen patients who experienced such sensations for years after amputations.

As regards *substitutes for limbs*, much depends upon the worldly position of the patients, and what pecuniary means they possesss, not only for their acquirement but also for keeping them in order and renewing them, for all these mechanical appliances wear out and sometimes get broken, while a skilful instrument maker and the necessary money are not always at hand for putting them in order again.

Artificial arms and well imitated hands are matters of adornment and luxury. Active movements of the fingers cannot be attained, but certain mechanical appliances for taking hold of objects may be employed, including springs to be opened with the other hand. I shall not go into further details on this point here. For working men, a leather case is made which is placed about the stump of the forearm or arm and fastened to it with straps. To the lower part of this case a strong piece of wood is attached, into which hooks, rings, &c., can be secured according to the occupation of the patient. On Sundays he screws on a hand carved out of wood. It appears incredible how much intelligent persons can effect with such simple appliances. I have in my possession a long calligraphic letter from a man both of whose hands I was obliged to amputate; he was an engineer and was so unfortunate as to have both his hands caught in the wheel of a water-mill and almost torn off. He afterwards earned his living as a writer! What irony of Fate!

As regards the lower extremities, there are few stumps upon which the patient can stand so as to rest the whole weight of the body upon them. These are the stumps after amputations and exarticulations about the foot and ankle. In many cases the stump

after exarticulation of the knee also is capable of supporting the weight of the body. In other cases the patients do not lean upon the amputation-stumps but upon the condyles of the tibia and upon the tuber ischii, which bony parts are supported from below by a well-cushioned firm ring which forms the upper end of the case of the artificial foot into which the stump is introduced. After amputations of the leg, it is desirable that the weight of the body should be distributed equally to both the parts mentioned. Another method is to let the patient rest the bent knee, in case of amputation of the leg, upon a wooden leg, in which case all idea of moving the knee-joint is given up. I will not go further here into details concerning the construction of artificial limbs, and will only remark that a certain amount of dexterity and intelligence is necessary for the use of them, as well as the pecuniary means for the repairs more or less frequently required. For the working classes, with whom we have chiefly to deal in hospitals, it is much more practical to use firm wooden legs, and many members of the higher classes, who have been inconvenienced for years by complicated appliances, eventually have recourse to them. The mode of progression in the two cases is so different that any one who has been accustomed for years to the use of the simpler apparatus requires much perseverance and a complete technical understanding of the complicated one to enable him to accustom himself to it.

Simple as the operations of amputation and exarticulation now appear to us, it cannot be denied that from the time of Hippocrates until the most recent period constant advances have been made. That large portions of the extremities may be lost without danger to life was proved first of all by the spontaneous separation of gangrenous parts; the first amputations were performed for the removal of such gangrenous limbs, and the bones were sawn through in the gangrenous part, or at the line of demarcation. More numerous indications for amputations were accepted very slowly at first, and one special obstacle to the development of this operation was the circumstance that the hæmorrhage could not be controlled with certainty. Styptics and the ferrum caudens might suffice for the leg and forearm, but no further. The development of operations for amputation depended, therefore, upon the advance made in the methods of checking the hæmorrhage; not until after the general introduction of ligatures and the invention of the tourniquet could the greater amputations be ventured upon. The method of ampu-

tating limbs by cutting them off by means of a ligature was first carried out by Guy de Chauliac, and further developed afterwards by Ploucquet. These methods of amputation have again been attempted quite recently, and écrasement (Chassaignac), galvano-caustic (v. Bruns), and the elastic ligature (Dittel) employed, but have met, on the whole, with little approval. Later on, the attention of surgeons was especially directed to the question how amputations could be performed most quickly, so as to cause as little pain as possible, and how the soft parts must be divided to avoid the formation of conical stumps. Now that we can lessen the pain by means of narcosis, and the loss of blood by the bloodless method, rapidity of operation in amputations and exarticulations is scarcely taken into consideration. Attention is directed exclusively to the formation of the stump, also since the beginning of this century to the establishment of conditions for healing by the first intention, and during some decennia to avoiding any infection from without or by the wound-secretions, and to the prevention of pyæmia, the most dangerous enemy of patients after amputations. These latter circumstances now occupy our attention chiefly, and the modern technology of these operations is directed especially to these important points.

The first method employed in the time of Celsius was a circular incision with drawing back of the skin. This gradually became developed further and further. Lowdham (1679) is generally regarded as the inventor of the one-flap operation; that method was then perfected by Verduin (1696). The oval incision was invented by Scoutetten. Ravaton and Vermale are said to have been the first to employ the two-flap operation. You will find a very exact sketch of the history of amputations, partly in that of operations generally by Sprengel, partly in the excellent theory of operations by v. Linhart, which I cannot recommend strongly enough to you.

It now remains for me to make some general observations on

Resections.

As I have mentioned already, the sawing, chiselling, and scraping out of diseased or injured pieces of bone from the diaphyses or bodies of the bones is called "*resection in the continuity.*" Most of the operations of this kind have been spoken of already in connection with compound fractures, necroses, and caries; also the

so-called operations of osteotomy for orthopædic purposes. You will have so many opportunities of seeing the technical details of these operations in the clinique that I shall not enter further into the subject here, which is, for the most part, very simple. The indications are furnished by what has been said formerly.

Of "*resections of the joints*" we have also spoken at an earlier period; I have told you already that these operations, which come into question in private practice in cases of caries especially, have different indications, a different prognosis, and different final results in connection with almost every joint. The same holds good for resection of joints after gunshot wounds; each joint has its own resection-history. Resections, especially total resections of joints, are much more modern operations than amputations. The first resection of a carious head of the humerus was performed by White in 1768, resection of the elbow-joint by Moreau in 1782, of the head of the femur by White in 1769, of the knee-joint by Park in 1762. These operations met with very little approval at first, however; they were declared to be too difficult and to require too much time, to be too painful therefore, while but little was expected from their final results. About thirty years ago only can resections of the joints be spoken of as operations accepted by surgeons generally, and the development of their technical details is constantly becoming more complete. It was only proposed at first to remove the diseased portions of bone without losing the extremity, so as to bring the diseased process to a healthy termination. Later on it was sought to render the function of the false joints formed after resections as perfect as possible, and to adapt thereto the direction of the incisions, the method of operating, and the after-treatment; some surgeons even went so far as to cut out stiff, healed joints to exchange them for movable false joints. Too favorable views were perhaps taken for some time of what may be attained by these operations, and the indications therefor extended too far, but striking results have certainly followed some of them, and with the concentrated attention now directed to many of them it may be expected that the indications, the technical details, the prognosis, and the after-treatment, will become more and more firmly established.

The incisions in cases of resection must be made in such a direction that none of the larger vessels and nerves and as few muscles as possible shall be injured, and yet space enough provided

to expose freely and saw off the ends of the bones. When these operations were first performed they appeared so difficult that it was thought necessary to lay open the joints very extensively with large deep flaps for the purpose of dividing conveniently the ligaments of the joints and the attachments of the muscles, and of exposing the ends of the bones to a considerable extent for the purpose of sawing through them. Later on, when more and more importance began to be attached to rendering resected joints as useful as possible, these operations were performed with more economy of the parts involved; care was taken not to cut through the tendons obliquely, or to make the wound in the skin larger than necessary. Eventually, as much as possible of the periosteum about the ends of the bones was preserved, and the attachments of the muscles were left in connection with it, while the periosteum itself was detached from the ends of the bones with the rasp, and thus operations were performed with the least possible injury to the vessels, and in the thickened tissues, in the case of chronic inflammatory processes, after which much less violent inflammatory and febrile reactions set in than after operations in perfectly healthy parts.

B. v. Langenbeck was the first to develop with perseverance the indications for resections of the joints, and is still occupied unceasingly in bringing the technical details of these operations to perfection. He also introduced especially the simple longitudinal incisions, which are now in general use for resections of the shoulder-, elbow-, and hip-joints; for the knee-joint an anterior flap-incision with a broad base superiorly is equally advantageous. Resections of the wrist- and ankle-joints are usually performed with two lateral longitudinal incisions.

The instruments now in general use for resections are, with the exception of the chain-saw (Jeffray), entirely those proposed by v. Langenbeck; strong knives with a blade 2—2·8 inches long and a thick back. These knives are introduced down to the bone at once and the incision in its whole depth made with one movement. The periosteum is pushed off the bones with broad or narrow, more or less bent, half sharp rasps, the ligaments of the joints and many of the attachments of the muscles only not always being separable in this manner, in which case they must be detached with the knife close to the bone, so that the ends of the bones may be entirely freed from soft parts. The ends of bone are then to be sawn off

with a chain, or pointed, or small amputation-saw, after having been firmly fixed with a bone forceps or hook and the soft parts held back with blunt double hooks. Sharp edges of bone are to be removed with cutting bone forceps.

The patient must be narcotised before the operation, the limb to be operated upon emptied of its blood and carefully cleaned. When the operation has been completed, the hæmorrhage must be checked most carefully, the wound washed out well with new clean sponges, and the limb then placed in a dressing which must be so arranged that the extremity is quite firmly fixed without undue pressure at any particular point, that the wound is free, and that there is no obstruction to the escape of the secretions. The position of the patient must, at the same time, be a comfortable one, and one that can be changed without any movement of the extremity within the dressing.

I have not found it beneficial, before the removal of the compressing ring placed above the wound, to plug the latter with lint and then apply a compressing dressing, because the whole dressing then becomes so thoroughly saturated with blood that it soon has to be renewed entirely. I prefer first checking the hæmorrhage completely by ligatures, acupressure, syringing out the wound with iced water, &c., before the dressing is applied; I then place drainage tubes dipped into glycerine or carbolic acid in the wound to carry off the secretions into vessels placed beneath the limb.

When possible, I apply a plaster-of-Paris bandage before the operation, make correspondingly large openings at the points at which I intend to perform it, then cut open the dressing on one side, and remove it for the purpose of operating, and thus have a plaster-of-Paris case which, when applied after the operation, will fit exactly. The extremity may be suspended by the aid of this plaster-of-Paris case, or it may be placed upon a Ris's splint. Other operators prefer previously arranged wooden or iron splints; there are many methods here for attaining the same results by means of the most various materials. After resection of the hip-joint, it is not usual to apply any dressing to the hip itself, but to cause extension by means of weights. I have also found Lister's method of treating wounds very useful in some cases.

Resection wounds are always rather complicated cavernous wounds, and never heal except by granulation and suppuration.

The healing process always requires a considerable time for its completion. This is, unfortunately, a great objection to resections in weakly, atrophic subjects, and in such persons it is, moreover, never certain whether the caries may not spread to contiguous portions of bone or on the sawn surfaces, and the wound assume an ulcerative character.

The shortest duration of the healing process after resections is probably two to three months. Indolent fistulæ often remain open for months or years.

Especial attention has been directed of late to the final results of resections of the joints. False joints after resections may, for instance, become so loose as not to admit of any active movement, and the resected limb hangs totally useless from the body. Other such joints allow a certain amount of active movement. Then follow the joints which have almost complete normal mobility, with normal muscles and power; lastly, joints which have become anchylosed, which are, at all events, more useful than false joints which admit of passive movement only. The amount of bony parts removed, the degree of regeneration of bone about the resected ends of bone, the care taken in dividing the attachments of muscles, and the natural muscular power of the patient, have great influence upon the final results. Gymnastic exercises, baths, electricity, the adaptation of suitable apparatuses, are all of importance for the attainment of favorable results. But since these results differ in the case of each joint, and require different methods and apparatuses for treatment, this can only be spoken of more exactly in connection with the resection of individual joints.

As regards the *prognosis quoad vitam,* the same holds good for resections of the joints as for amputations. Resections on acccount of caries generally run a more favorable course than resections on accounts of injuries. The danger increases in proportion as the joint is situated nearer to the trunk of the body.

LIST OF AUTHORS REFERRED TO.

† This sign signifies died.

Abernethy, John († 1831, in London)
Abulkasem († 1106)
Aeby, *Professor of Anatomy in Bern*
Alexander v. Tralles (525—605)
Alexandrian School
Albert, *Professor of Surgery in Innsbruck*
Amabile, *Professor in Naples*
Anders, *Surgeon in Livonia*
Anel, Dominic, *Surgeon in Turin* (commencement of eighteenth century)
Antyllus (third century)
Arndt, *Greifswald*
Arnold, J., *Professor of Pathological Anatomy in Heidelberg*
Asclepiades
Aseli (1581—1626)
Auerbach, *Breslau*
Avenzoar (1126)
Avicenna (980—1037)
Barbieri, *Surgeon, Vienna*
Bärensprung, von (1822—1864)
Bardeleben, *Professor of Surgery, Berlin*
Bartscher, *Surgeon in Westphalia*
Barwell, *Surgeon in London*
Baum, *Professor of Surgery in Göttingen*
Baynton, *English Surgeon*
Beck, *Surgeon-General, Baden*
Bell, Benjamin (1749—1806)
Bellocq, Jean (1732—1807)
Benecke, *Professor, Marburg*

Bergmann, *Professor of Surgery in Dorpat*
Bernard, Claude, *Professor of Physiology, Paris*
Bernhardt, M., *Surgeon in Berlin*
Biermer, *Professor, Breslau*
Biesiadecki, *Professor of Pathological Anatomy in Cracow*
Bilguer, John Ulrich (1720—1796)
Binelli
Bizzozero, *Professor of Pathological Anatomy in Padua*
Bollinger, *Professor, Munich*
Boinet, *Surgeon in Paris*
Bonnet, *Surgeon in Lyons*
Botalli (1530—1591)
Bouvier, *Surgeon in Paris*
Boyer, Baron (1747—1833)
Branca (fifteenth century)
Brasdor (1721—1799)
Breschet, G. († 1845)
Breslan (1829—1867)
Breuer, *Vienna*
Broca, *Professor of Surgery, Paris*
Brodie, Sir Benjamin (1783—1863)
Bromfield, William (1712—1792)
Brown-Séquard, *Physician in London*
Brücke, E., *Professor of Physiology in Vienna*
Bruns, von, *Professor of Surgery in Tübingen*
Bruns, P., *Tübingen*
Brunschwig, Hieronymus (born in 1430)
Bubnoff, *Military Surgeon, St. Petersburg*
Buhl, *Professor of Pathological Anatomy, Munich*
Burow, *Professor of Surgery, Königsberg* († 1864)
Celsus, Aulus Cornelius (35 A.C.—45 A.D.)
Chassaignac, *Surgeon in Paris* († 1869)
Cheselden, William (1688—1793)
Chrobals, *Vienna*
Ciniselli, *Surgeon in Milan*
Civiale (1792—1867)
Cohn, *Professor of Botany, Breslau*
Cohnheim, *Professor of Pathological Anatomy in Breslau*
Cooper, Sir Astley (1768—1841)
Cruveilhier, *Professor of Pathological Anatomy, Paris* († 1873)

Czerny, *Professor of Surgery in Freiburg*
Davaine, *Professor in Paris*
Delpech (1772—1832)
Desault, Peter (1774—1795)
Dieffenbach, John F. (1795—1847)
Dieulafoi, *Physician in Paris*
Dittel, *Professor of Surgery in Vienna*
Dolschenkow, *Russian Surgeon*
Dorsey, *Surgeon in Philadelphia* (1783—1818)
Doutrelepont, *Professor of Surgery in Bonn*
Dubois-Reymond, *Professor of Physiology in Berlin*
Duchenne, de Boulogne, *Physician in Paris*
Dumreicher, Baron v., *Professor of Surgery, Vienna*
Dupuytren, Baron (1778—1835)
Eberl, *Professor in Berlin* († 1872)
Eberth, *Professor of Pathological Anatomy, Zürich*
Ehrlich, F., *Surgeon in Vienna*
Eichhorst, *Surgeon in Königsberg*
Eschricht, *Professor of Anatomy in Copenhagen*
Esmarch, F., *Professor of Surgery in Kiel*
Estländer, *Professor of Surgery in Helsingfors*
Eustachio († 1579)
Fabricius v. Hilden (1560—1634)
Falopia (1490—1563)
Fick, Adolph, *Professor of Physiology in Würzburg*
Fischer, *Professor of Surgery in Breslau*
Flourens (1791—1867)
Fock, Carl (1828—1863)
Förster (1822—1865)
Follin (1823—1867)
Fox, Wilson, *Physician in London*
Frey, *Professor of Zoology in Zürich*
Frisch, *Professor of Anatomy, Vienna*
Froriep, Robert (1804—1861)
Galenus, Claudius (131—201)
Gersdorf, Hans v. (1520)
Goll, *Physician in Zürich*
Golubeir, *Russian Surgeon*
Golz, *Professor of Physiology in Strassburg*
Graefe, Carl von (1787—1840)

Graefe, Albrecht von, *Professor of Ophthalmology in Berlin*
Grawitz, *Surgeon in Schlesia*
Gross, *Professor of Surgery, Philadelphia*
Gruber, W., *Professor of Anatomy, St. Petersburg*
Guerin, *Surgeon in Paris*
Güterbock, *Surgeon in Berlin*
Guidode, Cauliaco (fourteenth century)
Gurlt, *Professor of Surgery in Berlin*
Gussenbauer, *Professor of Surgery in Luhbick*
Haller, Albrecht, von (1707—1777)
Hallier, *Professor of Botany, Jena*
Harvey, William (1578—1658)
Hebra, *Professor of Dermatology in Vienna*
Hecke, van, *Belgian Engineer*
Heiberg, *Surgeon in Christiania*
Heine, Bernhard, *Instrument Maker and Honorary Professor of Surgery in Würzburg; contemporary of Cajetan von Textor*
Heine, C., *Professor of Surgery in Prague*
Heinecke, *Professor of Surgery in Erlangen*
Heister, Lorenz (1683—1758)
Heitzmann, *Surgeon in New York*
Henke, *Professor of Anatomy in Tübingen*
Henle, *Professor of Anatomy in Göttingen*
Hennen, John († 1828)
Hering, *Professor of Physiology in Prague*
Hiller, *Surgeon in Berlin*
Hjelt, *Surgeon in Sweden*
Hippocrates (460—377 A.C.)
His, William, *Professor of Anatomy in Leipzig*
Hoffmann, F. A., *Physician in Berlin*
Howship, *English Surgeon*
Hueber, *Professor of Surgery in Greifswald*
Hufschmidt, *Surgeon in Sweden*
Hunter, John (1728—1793)
Hunter, William (his writings appeared between 1756 and 1807)
Hutchinson, *Surgeon in London*
Jackson, *Physician in Boston*
Jacobson, *Professor in Königsberg*
Jeffray,
Jobert de Lamballe (1799—1863)

Jochman, *Physician in Prussia* †
Kern, Vincent v. (1760—1829)
Key, Axel, *Professor of Pathological Anatomy in Stockholm*
Klebs, *Professor of Pathological Anatomy in Prag*
Kilian, *Professor of Obstetrics in Prag* †
Koeber, *Professor of Surgery in Bern*
Koehmann, *Surgeon in Strassburg*
Köberle, *Professor of Surgery in Strassburg*
Kolbe, *Professor of Chemistry in Leipzig*
Kölliker, *Professor of Anatomy in Würzburg*
König, *Professor of Surgery in Rostock*
Köster, *Professor of Pathological Anatomy in Bonn*
Krause, *Physician in Hannover*
Kühne, *Professor of Physiology in Heidelberg*
Kundrat, *Professor of Pathological Anatomy in Graz*
Laënnec (1781—1826)
Lambl, *Professor in Charkow*
Lanfranchi († 1300)
Langenbeck, Conrad J. Martin (1776—1850)
Langenbeck, Bernhard von, *Professor of Surgery, Berlin*
Langer, C. *Professor of Anatomy, Vienna*
Langhans, *Professor of Pathological Anatomy in Bern*
Larrey, Jean Dominique (1776—1843)
Laudien, *Surgeon in Strasburg*
Lawrence, Sir William (1783—1867)
Leber, *Professor of Ophthalmology in Göttingen*
Lebert, *Professor of Clinical Medicine in Breslau*
Leiber, *Instrument Maker in Vienna*
Leroy d'Etiolles (1798—1861)
Leube, *Professor of Medicine in Erlangen*
Leyden, *Professor of Medicine in Strasburg*
Liebermeister, *Professor of Medicine*
Liebreich, *Professor of Medicine in Berlin*
Linhart, von, *Professor of Surgery in Würzburg*
Lister, *Professor of Surgery in Edinburgh*
Loeffler, *Surgeon-General, Prussian Army* († 1873)
Lösch, *Surgeon in St. Petersburg*
Lorinser, *Surgeon in Vienna*
Lossen, *Professor of Surgery in Heidelberg*
Loth, *Surgeon in Vienna*

Lotze, *Professor of Philosophy and of Medicine in Göttingen*
Lowdham (1679)
Lücke, *Professor of Surgery in Strasburg*
Lukornsky, *Russian Military Surgeon*
Luschka, von, *Professor of Anatomy in Tübingen*
Maas, *Professor of Surgery in Breslau*
Malgaigne (1806—1865)
Martin, *Professor of Obstetrics in Berlin* († 1876)
Maslowski, *Professor in St. Petersburg*
Mathysen, *Dutch Military Surgeon*
Melkel, Hemsbach v. (1821—1856)
Menel, *Saxon Military Surgeon*
Menzel, *Surgeon in Trieste*
Meyer, Herrmann, *Professor of Anatomy in Zurich*
Meynert, *Professor of Psychiatric in Vienna*
Middeldorpf, *Professor of Surgery in Breslau*, 1824—1868
Minnich, *Surgeon in Venice*
Mondino de Luzzi (fourteenth century)
Monro, Alexander (1696—1767)
Morand, *French Surgeon*
Moreau (1782)
Morton, *Surgeon in Boston*
Mcsengeil, *Surgeon in Bonn*
Mott, Valentine (1785—1865)
Müller, Johannes (1801—1858)
Müller, Max, *Surgeon in Cologne*
Müller, W., *Professor of Pathological Anatomy in Jena*
Nassilow, *Teacher of Surgery, St. Petersburg*
Neudörfer, *Military Surgeon in Vienna*
Neumann, E., *Professor of Pathological Anatomy in Königsberg*
Neumann, J., *Professor in Vienna*
Niemeyer, Felix von († 1871)
Ollier, *Surgeon in Lyons*
Oribasius (326—403)
Orth, *Teacher of Pathological Anatomy in Berlin*
Panum, *Professor of Physiology in Copenhagen*
Paracelsus, Bombastus Theophrastus (1493—1554)
Paré, Ambroise (1517—1590)
Park (1762)
Pasteur, *Professor of Chemistry in Paris*

Rosenberger, *Surgeon in Würzburg*
Roser, *Professor of Surgery, Marburg*
Roux (1780—1854)
Rust, John Nepomuk (1775—1840)
Samuel, *Professor of Pathology in Königsberg*
Sattler, *Tutor at University of Vienna*
Scarpa (1748—1832)
Schiff, *Professor of Physiology in Geneva*
Schmidt, Alexander, *Professor in Dorpat*
Schneider, *Saxon Military Surgeon*
Schneider, *Surgeon in Königsberg*
Schönlein, Lucas (1793—1864)
Schüller, *Surgeon in Hannover*
Schuh, Franz (1804—1866)
Schultze, Max, *Professor of Anatomy, Bonn* († 1873)
Schüppel, *Professor of Pathological Anatomy in Tübingen*
Schwalbe, *Surgeon in Weinheim*
Schwann, Theodor, *Professor of Physiology in Luttich*
Scultetus (1595—1645)
Scoutetten, *Professor in Paris about* 1830
Senator, *Physician in Berlin*
Seutin (1793—1862)
Siebold, Carl Caspar, von (1736—1807)
Silvestri, *Surgeon in Vicenza*
Simon, *Professor of Surgery in Heidelberg* († 1876)
Simpson, Sir James, *Professor of Obstetrics in Edinburgh*
Sims, Marion, *American Gynæcologist*
Soborow, *Military Surgeon in Moscow*
Skutsch, *Surgeon in Schlesia*
Sprengel, Kurt (1766—1833)
Stanley (1791—1862)
Steudener, *Teacher of Pathological Anatomy in Halle*
Störk, *Professor in Vienna*
Stricker, Solomon, *Professor of Pathology in Vienna*
Stricker, *Surgeon in Frankfort*
Stromeyer, Louis (*formerly Surgeon-General, Hannoverian Army*, † 1876)
Stromeyer, *Surgeon in Göttingen*
Súsrutas (first century)
Sydenham (1624—1689)

Syme († 1869, *in Edinburgh*)
Szymanowski, *Professor of Surgery in Kiew* († 1868)
Taylor, *Surgeon, of New York*
Textor, Cajetan von (1782—1860)
Theden (1714—1797)
Thiersch, *Professor of Surgery in Leipzig*
Tillmans, *Teacher at the University of Leipzig*
Traube, *Professor of Medicine in Berlin* (1818—1876)
Troja, Michael (1747—1827)
Tratula (twelfth century)
Tschausow, *Teacher of Surgery in Warsaw*
Valsalva (1666—1723)
Vanzetti, *Professor of Surgery in Padua*
Velpeau (1759—1867)
Verduin (1696)
Vermale, *French Surgeon* (eighteenth century)
Verneuil, *Professor of Surgery in Paris*
Vesalius, Andreas (1513—1564)
Vezin, *Surgeon in Westphalia*
Vidal de Cassis, *formerly Professor of Surgery in Paris*
Villemin, *Surgeon in Paris*
Virchow, *Professor of Pathological Anatomy in Berlin*
Volkmann, Richard, *Professor of Surgery in Halle*
Wagner, A., *Professor of Surgery in Königsberg* († 1871)
Wagner, E., *Professor in Leipzig*
Waldenburg, *Professor of Medicine in Berlin*
Waldeyer, *Professor of Anatomy in Strassburg*
Waller, *English Physician*
Walther, Philip v. (1782—1849)
Wardrop, *English Surgeon*
Weber, Otto (1827—1867)
Wegener, *Teacher of Pathological Anatomy in Berlin*
Wells, Spencer, *Surgeon in London*
Wernher, *Professor of Surgery in Giessen*
Wertheim, *Physician in Vienna*
White (1769)
Winiwarter, A. v., *Teacher of Surgery in Vienna*
Wolff, J., *Surgeon in Berlin*
Würtz, Felix († 1567)
Wunderlich, *Professor of Medicine in Leipzig* († 1877)

Wutzer (1789—1860)
Wyss, O., *Professor in Zurich*
Wydozoff, *Russian Military Surgeon*
Zalessky, *Professor of Hygiene in Charkow*
Zeis, *Surgeon in Dresden* († 1868)
Zenker, *Professor of Pathological Anatomy in Erlangen*
Ziegler, *Physician in Würzburg*
Ziemssen, *Professor of Medicine in Erlangen*

INDEX.

CORRIGENDUM.

In Vol. II, p. 139, *read* "Reverdin," *instead of* "Keverdin."

PRINTED BY J. E. ADLARD, BARTHOLOMEW CLOSE.